A STUDY OF OBSERVATION IN TREATMENT MODALITY OF ACUTE AND CHRONIC POISONING CASES BY ANALYSING CLINICAL LABORATORY PARAMETERS AND ITS OUTCOME IN TERTIARY CARE HOSPITALS.

A Dissertation Submitted to

THE TAMIL NADU Dr. M.G.R. MEDICAL UNIVERSITY

CHENNAI- 600 032

In partial fulfilment for the requirements for the award of the Degree of

MASTER OF PHARMACY

IN

BRANCH-VII - PHARMACY PRACTICE

Submitted by

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Under the guidance of

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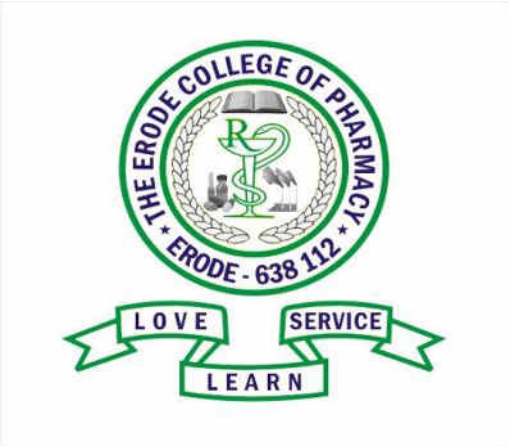

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This is to certify that the dissertation work entitled **"A study of observation in treatment modality of acute and chronic poisoning cases by analysing clinical laboratory parameters and its outcome in tertiary care hospitals"** submitted by **Register No: 261440411** to The Tamil Nadu Dr. M.G.R Medical University, Chennai, in partial fulfilment for the degree of **Master of Pharmacy in Pharmacy Practice** is the bonafide work carried out under the guidance and direct supervision of **Prof. Dr. J. NANDHAKUMAR, M.Pharm., Ph.D.,** Head, Department of Pharmacy Practice, The Erode College of Pharmacy and Research Institute, Erode- 638112, during the academic year 2015-2016.

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This is to certify that Mr. Muhammed Anas K.P. student of 'The Erode College of Pharmacy' has done a six month project (January 2016 to June 2016) as per the curriculum requirement of M. Pharm second year, in Critical Care Department of our hospitals.

He has completed the project "A Study of Observation in Treatment Modality of Acute and Chronic Poisoning Cases by Analyzing Clinical Laboratory Parameter's And Its Outcomes in Tertiary Care Hospital" satisfactorily.

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DECLARATION

The research work embodied in this dissertation work entitled **"A study of observation in treatment modality of acute and chronic poisoning cases by analysing clinical laboratory parameters and its outcome in tertiary care hospitals"** was carried out by myself in the Department of Pharmacy Practice, The Erode College of Pharmacy and Research Institute, Erode, under the guidance and direct supervision of **Prof. Dr. J.Nandhakumar, M.Pharm., Ph.D.,** Head, Department of Pharmacy Practice, The dissertation is submitted to **The Tamil Nadu Dr. M.G.R Medical University, Chennai,** as a partial fulfilment for the award of degree of **Mater of Pharmacy in Pharmacy Practice** during the academic year 2015-2016.

This work is original and has not been submitted in part or full for the award of any other Degree or Diploma of this or any other university.

Place: Erode **Register No: 261440411**

Date:

EVALUATION CERTIFICATE

This is to certify that dissertation work entitled "**A study of observation in treatment modality of acute and chronic poisoning cases by analysing clinical laboratory parameters and its outcome in tertiary care hospitals**", submitted by **Register no: 261440411** to the Tamil Nadu Dr. M.G.R Medical University, Chennai, in partial fulfilment for the degree of **Master of Pharmacy** is a bonafide thesis work carried out by the candidates at the Department of Pharmacy Practice, the **Erode College Of Pharmacy and Research Institute, Erode-638112** and was evaluated by us during the academic year **2015-2016.**

1. INTERNAL EXAMINERS **2.EXTERNAL EXAMINERS**

3. CONVENER OF EXAMINATION

Examination Centre: The Erode College Of Pharmacy and Research Institute.

Date:

ACKNOWLEDGEMENT

The secret of success is undaunted order, motivation, dedication, confidence on self and above all the blessing of god; I bow in reverence to the almighty for bestowing upon me all his kindness that has helped me throughout the journey of my life. Success is an outcome of collaborating efforts aimed that achieving different goals. I hereby take this opportunity to acknowledge all those who have helped me in the completion of this dissertation work.

My sincere thanks go to my respected guide **Prof. Dr.J.NANDHAKUMAR, M.Pharm., Ph.D.,** Head, Dept. Of Pharmacy Practice, The Erode College of pharmacy, for giving his valuable suggestions, guidance and constant encouragement throughout the project work.

I owe a debt of gratitude to our Principal **Dr. V.GANESAN, M.Pharm., Ph.D.,** for his support and for providing necessary facilities to carry out my project work successfully.

A special feeling of gratitude to my respected teachers **Prof. Mr. S.RAJARAJAN, M.Pharm.,** and **Prof. Dr. R.SENTHILSELVI, M.Pharm., Ph.D.,** Department of Pharmacy Practice who have played a key role in developing my interested and understanding Pharmacy Practice.

I take this valuable time to express my sincere thanks to **Prof. Dr.N.GANAPATHY, M.B.B.S, D.A, M.D, F.C.C.P, D.C.C.M, MC.C.A., Director** of Dhanvantri Critical Care Centre, Erode. for providing his valuable time and advices. Which help me to materialized this project in most perfect manner.

I express my special thanks to **Prof. Bhopathi Mahibalan, M.Sc., M.Phil.,** and **Lab incharge** in Dhanvantri Critical Care Centre, Erode. Who had taken the pain to provide me with all the facilities for the completion of my project work.

I express my sincere thanks and respectful regard to our beloved President **Dr. KR.PARAMASIVAM, M.Sc., Ph.D.,** and the Secretary & Correspondent **Shri. A. NATARAJAN, B.A., HDC** Treasure **Lion. R. KOLANDAISWAMY** for all facilities that were provided to me at the institution for enabling me to do the work of this magnitude.

It gives me an immense pleasure to express my deepest than heartfelt, ineptness and regards to **Prof. Dr. J. NANDHAKUMAR, M.Pharm., Ph.D.,** who provided me much of the stimuli in the form of suggestions, guidance and encouragements at all stages of my work. His critical evaluation and deep-rooted knowledge and strive for excellence will always remain a source of inspiration to me.

My special thanks go to **Mr. K. VARADHARAJAN, B.A., M.L.I.S**., Librarian, for providing me the library facilities. My sincere thanks go to all teaching and non-teaching staff of The Erode College of Pharmacy and Research Institute, who gave a helping hand to me while carrying out this study.

I expressed my gratitude to all **Teaching and Non-Teaching Staff** of The Erode College of Pharmacy & Research Institute, Erode.

The project would not be a resplendent one without the timely help and continuous support by ever loving friends **Mr. DANISH T.K, Mr. MUHAMMED SHABEER A, Mr. K. TAMILARASAN, Miss. MEERA NADHINI,** for their support and encouragement throughout the study.

Last but not least I express my warmest and warm and most important acknowledgement to my parents and family **Mr. MOHAMMED AKBAR K.P, Mrs. ASMABI K.,** and my brothers **Mr. ARSHAD K.P and Miss. AFIDHA K.P** with deep appreciation and moral support encouragement and everlasting love that served as a source of my inspiration, strength, determination and enthusiasm at each and every front of my life, to transfer my dreams in to reality.

Register No: 261440411

ABBREVATIONS

AlP	**-**	**Aluminum Phosphide**
OPCP	**-**	**Organo Phosphorous Compound Poisoning**
OP	**-**	**Organo Phosphorous**
CO	**-**	**Carbon Monoxide**
Zn_3P_2	**-**	**Zinc Phosphate**
DMS	**-**	**Delayed Neurophychiatric Syndrome**
COPP	**-**	**Chronic Organo Phosphorous Poisoning**
AOPP	**-**	**Acute Organo Phosphorous Poisoning**
BUN	**-**	**Blood Urea Nitrogen**
PaO_2	**-**	**Partial Pressure Arterial Oxygen**
O_2	**.**	**Oxygen**
H_2S	**-**	**Hydrogen Sulphide**
HCO_3	**-**	**Bicarbonate**
AAS	**-**	**Atomic Absorption Spectroscopy**
NAA	**-**	**Neutron Activating Analysis**
QRS	**-**	**Quatron Resonance System**
Na	**-**	**Sodium**
Ca	**-**	**Calcium**
P	**-**	**Phosphorous**
K	**-**	**Potassium**
H_2S0_4	**-**	**Sulphuric Acid**
CAVH	**-**	**Continuous Arterio-Venous Hemofiltration**
CNS	**-**	**Central Nervous System.**

CVVH - **Continuous Veno- Venous Hemofiltration**

CLIA - **Chemiluminescence Immune Assay**

ECG - **Electro Cardiograph**

FPIA - **Fluorescence Polarization Immune Assay**

HPLC - **High Performance Liquid Chromatography**

ICP-AE - **Inductively Coupled Plasma –Atomic Emission Spectroscopy**

I.M - **Intra Muscular**

ISE - **Iron Selective Electrode**

I.V - **Intra Venous**

LSD - **Lysergic Acid Diethylamide**

MRI - **Magnetic Resonance Pattern**

RIA - **Radio Immune Assay**

SC - **Sub Cutaneous**

TLC - **Thin Layer Chromatography**

UTI - **Urinary Tract Infection**

WHO - **World Health Organization**

OPP - **Organophospherous Poisoning**

EDB - **Ethyl Di Bromide**

RFT - **Renal Function Test**

RR - **Respiratory Rate**

PR - **Pulse Rate**

ET - **Endotracheal**

hRf - **Retention Factor**

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ABSTRACT

BACKGROUND

Poisoning cases is a significant contributor to morbidity and mortality all over the world. Acute poisoning cases are the most common causes of emergency hospitalization. Poisoning cases were mainly diagnosed by victims from patients and biochemical, hematological test and also using the qualitative and quantitative analytical techniques in the toxicology department.

PATIENTS & METHODS

The study was conducted at Govt. Head Quarters Hospital Tirupur-642206, Govt. Head Quarters Hospital Erode-638112, Dhenvendhri Critical Care Hospital, karungal palayam Erode-638003. It was a retrospective observational study, observation were made on the basis of patient's histories such as age, sex, mode of poisoning, nature of poisoning, diagnosis method used for the identification of compound, and treatment given to the patients.

RESULT AND ANALYSIS

From this retrospective study was observed that, there is an alarming increase of poisoning cases is mainly for suicidal purpose (69%). Agricultural poisons were found to be the most common causes of acute poisoning cases (55%). The major number of poisonings cases are reported in the young people's age range (26-35 years old) (35%) and followed by the peoples age range (15-25years old) (22%). Males were the major victims in overall poisoning cases (61%) and the most poisoning cases were from rural area than the urban area.

CONCLUSION

Agricultural poisons was found to be the most common causes of acute poisoning, Aluminum Phosphate, Monochrotophos, Zinc Phosphate, Snake bite, Oleander poisoning, Cow Dung Powder poisoning were the most common causes of acute poisoning. Most of the treatments were provided to the patients according to symptomatic treatment only, based on the evidence or sample of poisons brought with patients.

INTRODUCTION

Poison is a substance that causes damage or injury to the body and endangers one's life due to its exposure by means of ingestion, inhalation or contact. It's also a medicine in a toxic dose [1]. Poisonings and snake bites constitute a major cause of hospitalization and mortality in developed as well as developing nations [2].According to WHO data, in 2012 an estimated 1,93,460 people died worldwide from unintentional poisoning of these deaths, 84% occurred in low-and middle income countries. In the same year, unintentional poisoning caused the loss of over 10.7 million years of healthy life [3].

Nearly a million people die each year as a result of suicide, and chemicals account for a significant number of these deaths. For example it is estimated that deliberate ingestion of pesticides causes 370,000 deaths each year. The number of these deaths can be reduced by limiting the availability of, and accesses to, highly toxic pesticides [3, 4].

Acute poisoning is defined as acute exposure (less than 24hrs) to the toxic substance. Acute poisoning due to accidental and suicidal exposure causes significant mortality and morbidity throughout the world [5].According to World Health Organization (WHO) globally more than 3million of acute poisoning cases with 220,000 deaths occur annually. (WHO 1999) It has been estimated that, in India five to six persons per lakh of population die due to acute poisoning in every year [6].Poisoning is the fourth common cause of mortality in India. Pattern of poisoning in a region depends on various factors include availability and access to which the poison, socioeconomic status of an individual, cultural and religious influences, etc [2, 6].

Rapid industrialization, introduction of newer range of drugs for treatment and massive use of pesticides in agriculture has increased the incidence of poisoning. Acute poisoning is a worldwide problem and could be intentional or unintentional. The unintentional or accidental poisoning is common among children and contributes to increased childhood morbidity and mortality [7]. Children get poisoned accidentally because of their exploratory nature and their desires to imitate adults. Adults do also get poisoned through intentional poisoning that could be a suicidal attempt. Poisoning can occur as a result of a wide range of causative agents. Those include chemicals such as cleaning agents, cosmetics and other household products [8, 9].

Medications are also other major causative agents of poisoning in many countries and are expected to be available in every house [10]. Pesticides are more prevalent in agricultural countries and are encountered in many acute poisoning cases. Animal envenomations are a problem in many areas of the world that are poisonous snakes, bees, Insects, spiders and scorpions etc. [11].

- **Poisons Are Classified According To the Mood of Poisoning [12].**

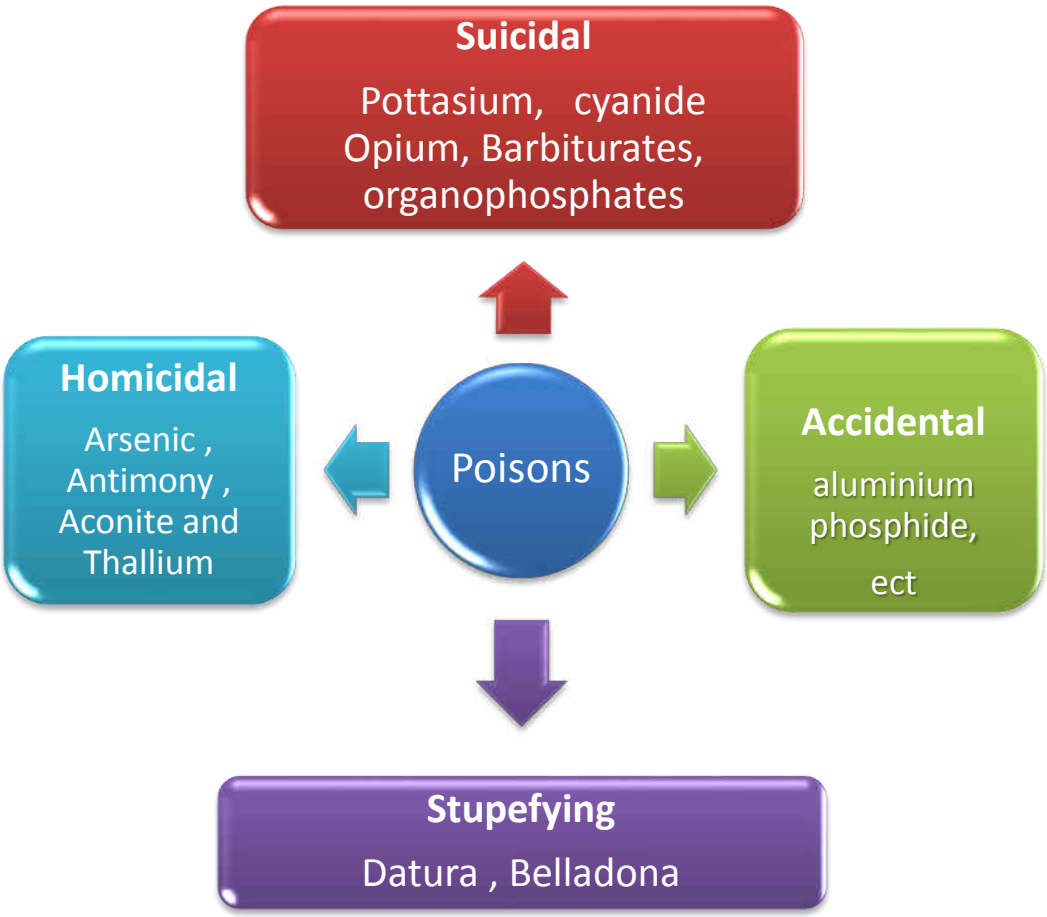


Figure 1: Poisons are classified according to the mood of poisoning.

- **Poisons Are Classified According To Mode of Action [12].**

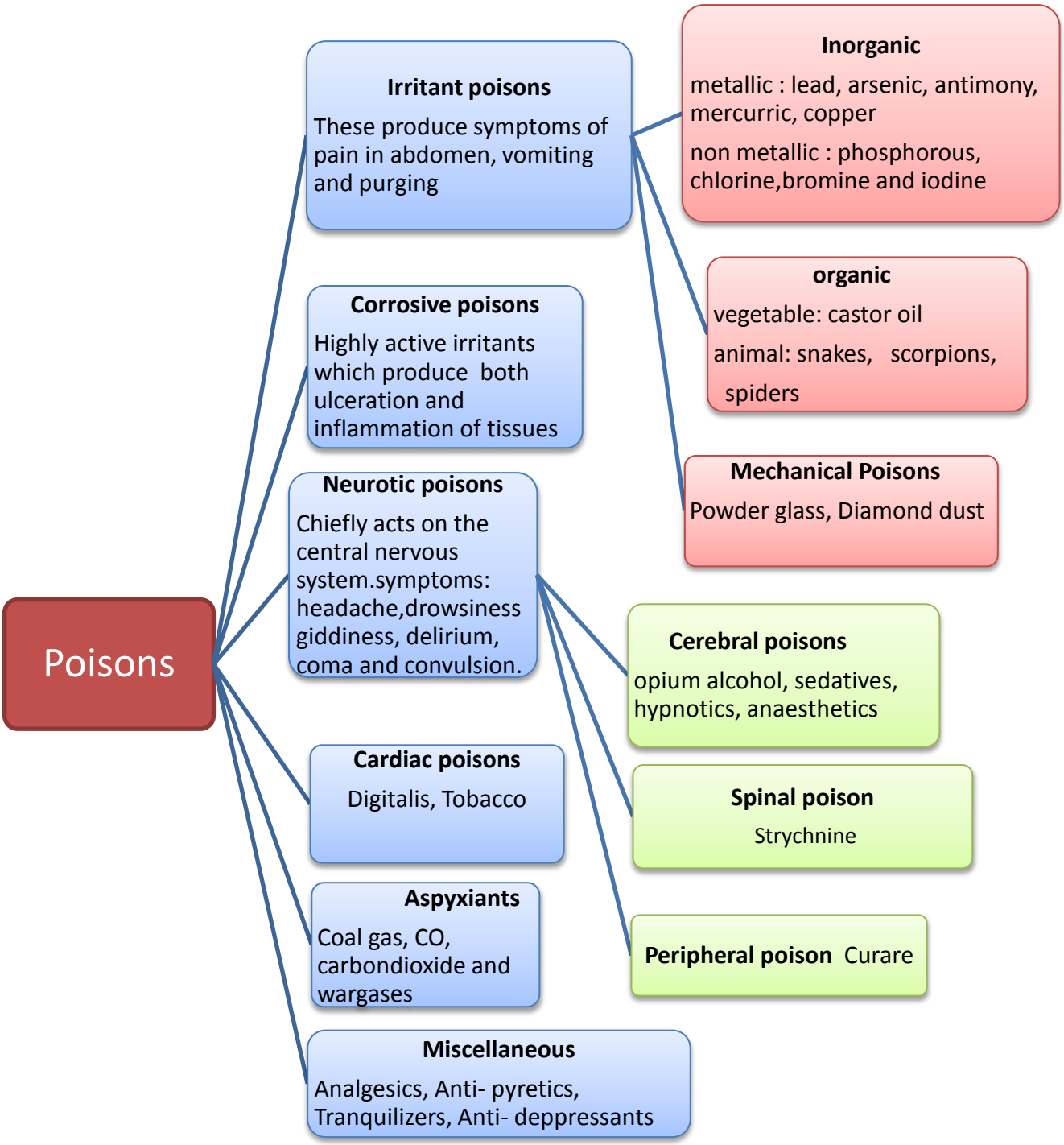


Figure 2: Poisons are classified according to Mode of action.

Table 1: Poisons are classified according to Source [13].

➢ DRUGS	**AMOEBICIDES** Carbarosone, Pentamidine, Ronidazole, Emetine..etc **ANAESTHETICS** Ketamine, Benzocaine, Lidocaine, Enflurane….etc. **ANTI-CONVULSANT** Beclamide, Carbamazepine, Lacosamide, Valproic acid….etc. **ANTI-BIOTICS** All preparations and their salts, Avopracin, Ertapenam sodium, Teicoplannin, Telithromycin….etc **ANTI- LEPROTICS** Clofazimine, Dapsone, Thiambutasine ….etc. **NSAID'S** Alclofenac,Azapropazone,Butorphanol,Celecoxib,Diclofenac, Etodolac,Flufenamic acid, Ibuprofen, Indomethacine, Nalbufine…etc. **ANTI -ASTHMATICS** Aminophyline, Etophyline, Doxophyline, Formoterol, Salbutamol etc. **ANTI -CHOLINERGIC** Atropine,Belladona,Benzhexol,Benztropine,Cyclopentolate,Dicyclomine,Ethopropazine,Fenpipramide,Fesoteradine,Glimepiride,Glycopyrrolate,Homatroine,Ipratropium…etc. **ANTI-DEPRESSANTS** Agomelatine,Bupropion,Citalopram,Deanol,Escitalopram,Fluoxetine,Hydrazines,Phenoxyethyl,Imipramine,Iproclozide,Maprotiline,Milnacipram,Mirtazapine,Nomifeneine,Phenelzine etc. **ANTI-DIABETICS** Actohexamide,Carbutamide,Chlorpropamide,Dapagliflozin,Exenatide,Gliclazide,Glimepiride,Lixisenatide,Metformin,Miglitol,Rapaglinide..etc **ANTI-HISTAMINES** Antazoline, Bilastine, Cetrizine, Doxylamine, Emedastine, Loratadine…etc. **ANTI-HYPERTENSIVES** Alseroxylon, Aliskiren, Azilsartan, Benazepril…etc.

➢ METALS	• Aluminium,Arsenic,Beryllium,CadmiumCopper,Iron, Lead,Lithium,Manganese, Mercury, Silver, Thallium, Tin, Zinc..etc
➢ FOOD MATERIALS	• Dairy products such as cheese, raw meat, chicken, Contaminated foods, bread, coconut milk etc.
➢ BITES/STINGS	• Bees, spider, snake, Scorpion
➢ HOUSEHOLD PRODUCTS	• Acids, alkalis, camphor, carbon monoxide, bleach, drain cleaner, rug cleaner, wallpaper cleaner, Glass, laundry ink, disc batteries, moth balls, cosmetics, essential oils, detergents.
➢ GARAGES	• Kerosene/paraffin, fire lighters, fire starting tablets, fire extinguishers, paints, painting supplies, Slug, weed killers, pellets, petrol, arsenic, lead, swimming pool chemicals, antifreeze, fumigants ,acids, alkalis, camphor, carbon monoxide, bleach, drain cleaner, rug cleaner, wallpaper cleaner, laundry ink, disc, batteries, moth balls, cosmetics, essential oils, detergents, car cleaning products, hobby chemicals
➢ INSECTICIDES	• Pyrethroids, organophosphorus, carbamates, Organochlorine, manganese compounds.
➢ RODENTICIDES	• Warfarines, indanodiones
➢ INSECT REPELLANTS	• Diethyltoluamide.
➢ HERBICIDES	• Bipyridyls, Chlorophenoxy glyphosate, Acetanilides, Triazines
➢ FUNGICIDES	• Thiocarbamates, Dithiocarbamates, Cupric salts, Tiabendazoles,Triazoles, Dicarboximides, Dinitrophenoles, Organotincompounds, Miscellaneous.
➢ FUMIGANTS	• Aluminium &Zinc phosphides, Methyl bromide, Ethylene dibromide.

➢ PESTICIDES	• Aldrin, Dieldrin, Chlordane, DDT, Endrin, Heptachlor Mirex, Toxaphene
➢ PLANTS	• Oleander, Poison ivy, Mushrooms, Thorn apple, Datura, Belladona,
➢ INDUSTRIAL& LABORATORY POISONS	• Acetic anhydride, N-acetylanthranilic acid , chloroform, Acetyl bromide, Acetyl chloride, Ammonia, Anthranilic acid, Formaldehyde, Hydrochloric acid,nitric acid.

- **Mechanisms of action of poisons are** [13, 14].

1. ***Local action***-Poisons act directly on the tissues and cause corrosion, irritation and inflammation.

2. ***Remote action***-As the poison gets absorbed systemically. It produces both specific CNS, spinal cord cardiac and nonspecific shock.

- **Factors modifying the action of poisons**

1. Dose

Small dose usually produce no toxic effects whereas large doses produce toxic effects on the body. Some individuals also exhibit phenomena like idiosyncrasy, allergy and synergism. The presentations are different with single or chronic exposure and with frequency of exposure.

2. Form of poison

(a) ***Physical state***- Gases and vapor's act more quickly than fluid poisons because they are absorbed immediately. Fluid poisons act faster than solid ones.

(b) ***Chemical combination***- Some substances in certain combination become inert like nitric acid and hydrochloric acid and certain other combinations becomes poisonous like lead carbonate and copper sulphide.

(**c**)***Mechanical combination***- The action of a poison is considerably altered when combined mechanically with inert substances.

3. Method of administration

A poison acts most rapidly when inhaled in gaseous or vapors form or when injected I.V followed by I.M. /S.C. and least rapidly when swallowed.

1. Condition of the body

(a) ***Age-*** Children is more susceptible than adults to toxins. In old age poisons have greater effects.

(b) ***Sleep and intoxication***-The bodily functions are lowest during sleep, so the poisons are absorbed slowly during sleep.

PHYSICAL EXAMINATION [13, 14, 15].

- Evaluation of Airway patency, Respiration, Circulation.
- Rapid assessment of mental status, temperature, pupil size, muscle tone, reflexes, skin and peristaltic activity.
- Separate patients into two groups
  - Depressed Status
  - Agitated Status
    - ✓ Drugs causing the depressed status are sympatholytic like adrenergic blockers, anti- arrhythmic, anti-hypertensive, anti-psychotics and cyclic antidepressant. Cholinergic like nicotine, carbamates, organophosphates, physostigmine, pilocarpine; Sedative-hypnotics like alcohols, barbiturates, benzodiazepines; Narcotics like analgesics, anti-diarrheal agents and others like CO, cyanide, H_2S, hypoglycemic agents, lithium and salicylates.
    - ✓ Drugs causing an agitated status are sympathomimetics like adrenergic agonists, amphetamines, caffeine, cocaine, ergot alkaloids, MAO inhibitors, theophylline. Anti-cholinergic like anti-histamines anti-parkinsonian drugs, anti-psychotics, anti-spasmodic, cyclic antidepressant, cyclobenzaprine; Drug withdrawal like a blockers, clonidine, ethanol, opioids, and sedatives-hypnotics. Hallucinogens like LSD, marijuana, mescaline, phencyclidine; and others like thyroid hormones.

- Pupil size and reaction to light, with the patient's physiological status gives a rapid clue regarding dominant ingestion.
    - Pinpoint pupils with agitation - Phencyclidine intoxication
    - Pinpoint pupils with lethargy - Narcotic overdose.
    - Dilated pupils not reacting to light – Anti-cholinergic intoxication.
    - Dilated pupils reacting to light - Cocaine intoxication.
- Level of coma of all poisoned patients should be assessed and recorded according to the REED COMA.

Table 2: Level of consciousness (Reed coma scale) [13].

Level Of Consciousness (**Reed Coma Scale**).					
Stage	Conscious Level	Pain Response	Reflex	Respiration	Circulation
0	Asleep	Normal	Normal	Normal	Normal
1	Coma	Decreased	Normal	Normal	Normal
2	Coma	None	Normal	Normal	Normal
3	*Coma	None	None	Normal	Normal
4	+Coma	None	None	Abnormal	Abnormal

- Look for muscle and motor movement. Muscle rigidity with hyperthermia is characteristic of neuroleptic malignant syndrome, malignant hyperthermia and black widow spider bite.

➢ POISONOUS PLANTS [16, 17].

Some plants are harmless, some sting, scratch or are poisonous. Poisonous parts of majority of species were seeds, latex and root or root bark. Besides these poisonous parts of some plants were fruits, stem, bark, tubers or bulbs and sometimes whole plant also. Some plants causes poisoning to both human beings as well as livestock populations, while some causes poisoning to human being only.

Table 3: Poisonous plants:

No.	Name of Plant/Family	Common Name/Hindi Name	Toxic Parts	Toxic Constituents	Fatal Dose	Fatal period
1	***Abrusprecatorius*** (Fabaceae)	Rosary pea, Crab's eyes, Gunchi (Hindi)	Roots, seeds and leaves	Abrin, Abrine and Abrasine	1 - 2 seed or 90-120 mg/kg (Abrin)	3 – 5 days
2	***Aconitum napellus*** (Ranunculaceae)	Indian aconite, Monkshood and mithazahar (Hindi)	All parts especially Dried tuberous root	Aconitine, Pseudo Aconite, Indaconitine Bhikhaconitine, Picraconitine, and Aconine	1 - 2 gram (root) 1-2mg (Aconotine)	2 to 6 h
3	***Aesculushippocastanum*** (Hippocastanaceae)	Horse –chestnut, Conker	All parts especially seeds	Aescin and Aesculin	_	_
4	***Alocasiamacrorrhiza*** (Araceae	Giant taro, Elephant ear	All parts	Calcium oxalate crystals and toxic Proteins	1/30 to 1/15 of a grain	15 min
5	***Anamirtacocculus*** (Menispermaceae)	Indian berry or fish berry	Fresh fruit	Picrotoxin and Dihydro-picrotoxine	_	_
6	***Antiaristoxicaria*** (Moraceae)	Upastree, Antiaris	Leaves and bark	α- Antiarin	LD - 0.116 mg/kg *i.v.*α-Antiarin	2 – 3 days

7	***Atropa belladonna*** (Solanaceae)	Deadly nightshade	All parts	Atropine, Scopolamine, Hyoscyamine, and Belladonnine	120 mg (atropine) 30 mg (hyoscine)	24 h
8	***Calotropisgigentea*** (Apocyanaceae	Calotropis and madar, akdo(Hindi)	Juice and roots	Uscharin, Calotoxin, Calactin and Calotropin	0.12 mg/kg calotropin	12 to 24h
9	***Argemonemexicana*** (Papaveraceae)	Argemone and Sial-kanta (Hindi)	All parts especially seeds	Berberine, Protopine, Sanguinarine and Dihydro-Sangunarine	–	–
10	***Cannabis sativa*** (Cannabinaceae)	Indian hemp Hashish	Leaves & fruit shoots	Cannabin, Cannabinon and Cannabinol	10 gm/kg b.wt.- bhang, 8 gm-ganja, 2 gram–charas	5 – 8 Days
11	***Capsicum annum*** (Solanaceae)	Chillies and Mirch (Hindi)	fruit	Capsaicin and Capsicin		
12	***Cerbaraodollum*** (Apocynaceae	Dabur, pilikirbir (Hindi)	Fruit and seed	Cerberin, Cerberoside, Odollin.	Kernel of one fruit	1 - 2 days or more
13	***Cerberathevetia*** (Apocynaceae)	Yellow oleander and Pila Kaner	All parts especially leaves & fruits	Thevetin, Thevetoxin, Nerifolin, Peruvoside, Ruvoside and Cerberin	8 - 10 seeds, 15 - 20 g of root, 5 to 10 leaves	Depend upon Quantity
14	***Cinchona officinalis*** (Rubiaceae)	Cinchona	Bark	Quinine, Cinchonine and Cinchonidine	8 - 10 g	2 h to 2 days

15	***Citrulluscolocynthis*** (Cucurbitaceae)	Indian wild gourd or bitter apple, bitter cucumber	Fruit, Root and dried pulp	Colocynthin	1 - 2 gram Colocynthin	24 h to 2-3 days
16	***Cleistanthuscollinus*** (Euphorbiaceae)	_	Leaves and Bark	Cleistanthin	0.5 mg/kg (animals)	_
17	***Colchicum autumnale*** (Colchicaceae)	Meadow saffron		Colchicine	Similar to arsenic poisoning	Similar to arsenic poisoning
18	***Conium maculatum*** (Apiaceae)	Poison hemlock	All parts	Coniine and Methyl Coniine	1 cm piece of plant	
19	***Crotolariaspectabilis*** (Leguminosae	Jhunjhunia (Hindi)		Mono-crotaline	65 mg/kg (chicken	_
20	***Croton tiglium*** (Euphorbiaceae)	Croton oil seed and Jamal-gota (Hindi)	Seed and oil	Crotin-a toxal-bumine, Tiglinic acids, Crotonic acid and Crotonoside	4 - 6 seeds, 1 - 2 ml oil	6 h to 3 days
21	***Cytissusscoparius*** (Leguminosae)	Yellow broom	Seed, leaves and twigs	Cytisine and Sparteine	_	_
22	***Daturafastuosa*** (Solanaceae)	Thorn apple and datura	All parts especially seeds and fruit	Atropine, Hyoscyamine, Hyscine and Dutarin	0.6 - 1 gram	24 h
23	***Dieffenbachia*** **sp.** (Araceae)	Dieffenbachia, Dumbcane	All parts	Cyanogenic Glycosides and Calcium oxalate	_	_

24	**Cyanogenic Glycosides andCalcium oxalate**	Fox glove	Roots, leaves and seeds	Digitoxin, Digitalin Digitalein and Digitonin	15-30 mg (Digitalin) 4 mg (Digitoxin	1 h to 24 h
25	***Dioscoreahispida*** (Dioscoreaceae)	Karukandu (Hindi)	Tubers	Spiro Alkaloid Dioscorine	120 mg/kg on mice	_
26	***Erythroxylum coca*** (Linaceae)	Coke, snow	Leaves	Cocaine, Procaine, Butacaine and Dibucaine	1 - 1.5 g Cocaine (Oral)	15 min to 10 h
27	***Euphorbia helioscopia*** (Euphorbiaceae)	Sun spurge	Milky latex	Non-HaemolyticSaponin and Phasin	_	_
28	***Gloriosasuperba*** (Liliaceae)	Superb lily, Flame lily and Kalihari (Hindi)	Tubers and roots	Colchicine, Superbine, Gloriosine and Glucosine	_	_
29	***Hyoscyamusniger*** (Solanaceae)	Henbane and Ajwayan(Hindi)	All parts	Atropine, Hyoscine and Hyscyamine	125mg (hyoscyamine)	_
30	***Jatropamultifida*** (Euphorbiaceae)	Bherenda (Hindi)	foliage and fruits	Curcin	_	_
31	***Gossypium*sp.** (Malvaceae)	Kapas (Hindi)	Seed oil	Gossypol	2.57 g/kg (Rat)	_
32	***Lantana camara*** (Verbenaceae)	Lantana, bunch berry	Entire plant, especially the berries	Lantanine, Lancamarone and Lantadenes A,B	_	_
33	***Lasiosiphoneriocephalus*** (Thymeleaceae)	Rameetha (Hindi)	Stem, leaves and bark	Lasioside and Lasiocephatin	0.5 mg/kg roots (cats)	_

34	***Lathyrussativus*** (Fabaceae)	Gross pea, Khesari (Hindi)	Seeds	β-Aminopropionitrile, β-Cyanoalanine,2,4-diamino Butyric acid, Selenium and 3-N-oxalyl-2,3-Diamino propionic Acid	_	_
35	***Lobelia nicotiancefolia (Campanulaceae)***	Indian lobelia and deonal (Hindi)	All parts	Lobeline	10 mg (Lobeline), 3.75 gram(leaves)	30 min to a day
36	***Manihotesculenta*** (Euphorbiaceae)	Sakarkanda (Hindi)	Tubers	Cyanogenic Glycoside	_	_
37	***Manihotutilissima*** (Euphorbiaceae)	Cassava, Tapioca	Root and leaves	Linamarin- a Cyanogenic Glycoside	300 g(adult 125 g (child) fresh root	_
38	***Mucunaprurita*** (Leguminosae)	Velvet bean, Konch (Hindi)	Seeds	Mucunain, Serotonin	_	_
39	***Myristicafragrans*** (Myristicaceae)	Nutmeg, Mace tree	Seeds	Myristicin and Elemicin	_	_
40	***Neriumodorum*** (Apocynaceae)	White oleander and Kaner (Hindi)	All parts	Neriodorin, Neriodorein and Karabin	15 - 20 g root / 24 - 36 hours	_
41	***Thevetiaperuviana*** (Apocynaceae	Suicide tree	Seeds and milky juice	Thevetin A and Thevetin B	Kernel of 01 fruit, 2 leaves (child)	_

42	***Nicotianatabacum*** (Solanaceae)	Tobacco and tambaku (Hindi)	All parts except ripe seeds	Nicotine	60 - 100 mg nicotine or 2 gramtobacco	5 - 15 min
43	***Ochrocarpuslongifolius*** (Gutiferae)	Naagkesar	All parts	Surangin A and Surangin B	LD - 9 mg/kg surangin A and 1 mg/kg surangin B (Cats)	_
44	***Papaversomniferum*** (Papavaraceae)	Opium poppy and afim (Hindi)	Ripe and dried capsules, petals and seeds	Morphine, Narcotine, Codeine and Thebaine	2g(opium), 0.2g (morphine) And 0.5g (codeine)	2 to 6 h
45	***Parthenium hysterophorus*** (Compositae)	Carrot grass	Leaves and seeds	Parthenin	_	_
46	***Pegnumhermala*** (Zygophyllaceae)	Wild rue	All parts	Harmaline, Harmine, Harmane, Harmalol, Asicine, Vasicinone	Harmaline-120 mg/kg (rat), Harman-200 mg/kg(rabbit)	
47	***Prunusamygdalus*** (Rosaceae)	Almond, Baadam (Hindi)	Almond	Amygdalin	20 almonds (adult) 10 almonds (Child)	

48	***Ricinuscommunis*** (Euphorbiaceae)	Castor bean, Erandi	Entire plant especially seeds	Recine and Recinine	6 mg of ricin (10 seeds)	2 to several days
49	***Solanumnigrum*** (Solanaceae)	Black nightshade	Immature berries	Solanine and Steroids		
50	***Strychnosnux-vomica*** (Loganiaceae	Poison nut and Kuchila	All parts especially seeds	Strychnine, Brucine and Vomicine	15 – 20 mg/kg (1 seed - Oral)	1 – 2 h

POISONOUS PLANTS IN INDIA.

POKEWEED (PHYTOLACCA AMERICANA)

Figure 3: Pokeweed (Phytolacca Americana).

SOLANUM SPECIES

Figure 4: Solanum species.

CHINA BERRY

Figure 5: Poisonous Plant China Berry.

CNS TOXINS

(Anti- cholinergic plant contain nicotinic alkaloid)

DATURA SPECIES

LEAVES OFFSET STEM

DATURA FLOWERS

Figure 6: Poisonous Plant Datura (ummetai)

ANGEL'S TRUM (BRUGMANSIA SPECIES)

Figure 7: Poisonous plant Angel trum

ANTI -CHOLLINERGIC PLANTS

TOBACCO PLANTS

Figure 8: Poisonous Tobacco plants.

HALLUCINOGENIC PLANTS

Figure 9: Hallucinogenic plants.

MYRISTICA FRAGRANS NUTMEG

Figure 10: Myristica fragrans nutmeg.

STRYCHNOUS NUXVOMICA

Figure 11: srtychnous nuxvomica.

CARDIAC GLYCOSIDES

Figure 12: Cardiac glycosides.

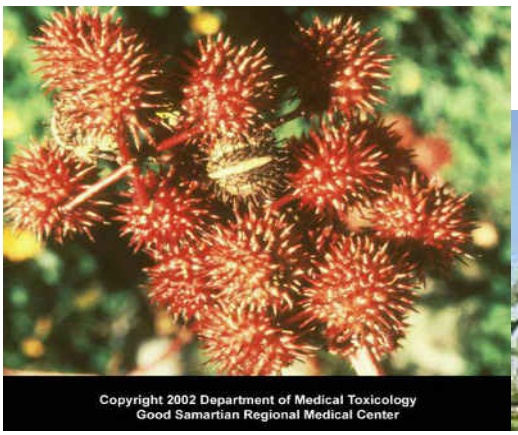
CARDIAC GLYCOSIDES (RODENTICIDE)

RHODODENDRON SPECIES
(Leaves are toxic)

Figure 13: Rhododendron species.

ACONITE (All part of the plant is toxic)

Figure 14: Aconite.



CASTOR BEANIT CONTAIN TOXIC ALBUMIN

Figure 15: Beanit & Contain toxin albumil.

ANTI –MITOTIC TOXINS

Figure 16: Anti-mitotic toxins.

RHUBARB(Leaf blades are toxic)

Figure 17: Rhubarb (leaf blades are toxics).

SNAKE BITE

A Snake will sometimes bite in self-defense if disturbed or provoked. Some snakes are venomous & can inject venom (Toxin) as they bite. A bite from a venomous snake is a medical emergency as they can be deadly if not treated quickly [18].

Snake-bite is a largely unrecognized public health problem that presents significant challenges for medical management. While reliable data are hard to obtain, it has been estimated that about 5 million snake-bites occur each year, resulting in up to 2.5 million envenoming, at least 100,000 deaths and around three times as many amputations and other permanent disabilities [3].

- **SYMPTOMS OF SNAKE BITE [18, 19].**

  - ✓ Pain, redness& swelling in the area of the bite.
  - ✓ Nausea (feeling sick) & vomiting.
  - ✓ Dizziness fainting.
  - ✓ Blistering &eventually, Gangrene in the area of the bite.
  - ✓ Shock
  - ✓ Muscle paralysis (An inability to move one or more muscles of the body) leading to difficulties, swallowing& breathing.
  - ✓ Bleeding.
  - ✓ Swelling of the lips gums & tongue.
  - ✓ Irregular heartbeat.

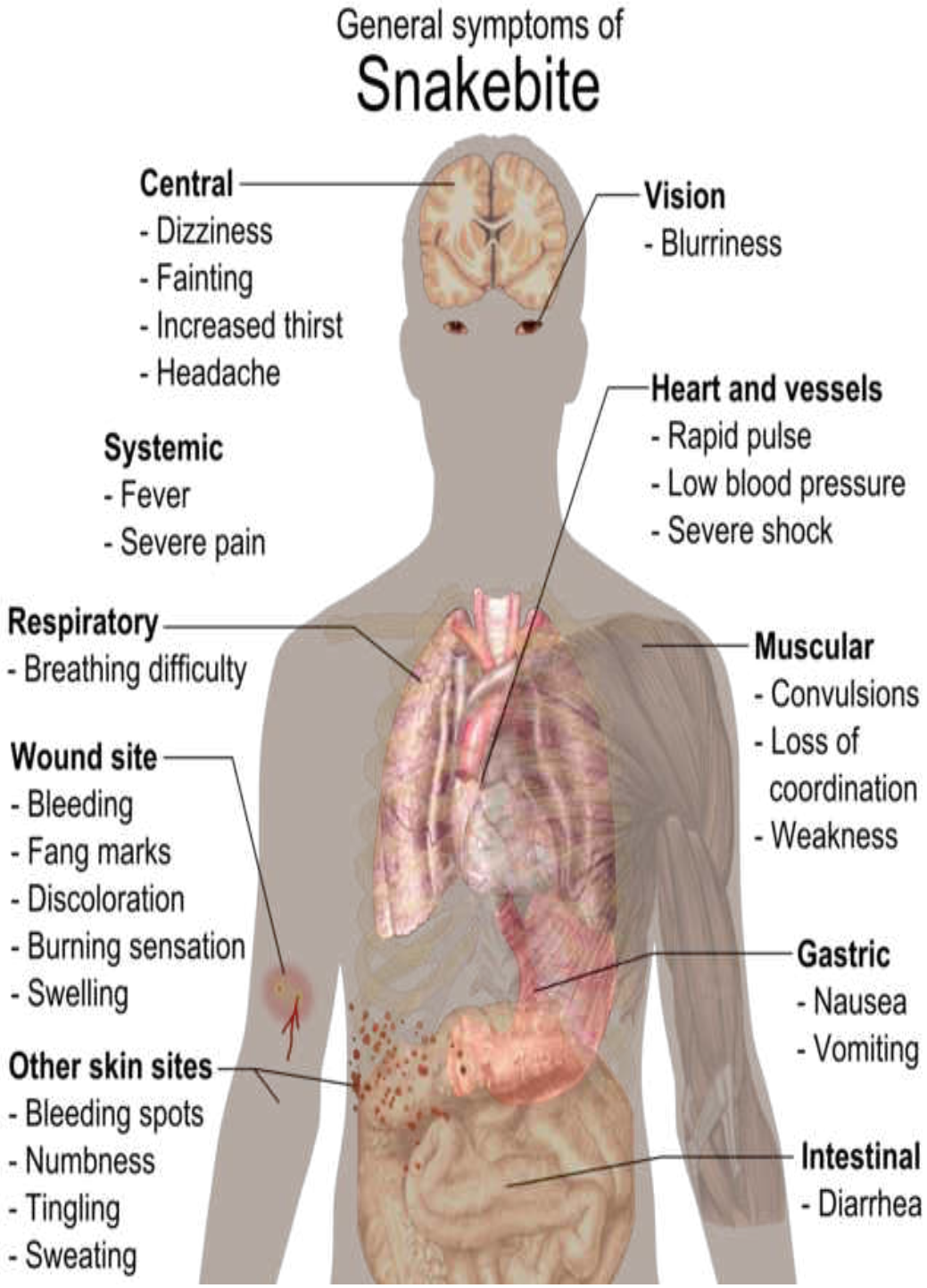


Figure 18: General symptoms of snakebite.

❖ POISONOUS SNAKES IN INDIA

KING KOBRAINDIAN COBRA

INDIAN KRAIT MALABAR PIT VIPER

RUSEEL'S VIPER SAW SCALED VIPER

Figure 19: Poisonous snakes in India.

MANAGEMENT OF SNAKE BITE [20]

- First aid treatment.
- Transport to hospital.
- Rapid clinical assessment and resuscitation.
- Detailed clinical assessment and species diagnosis.
- Investigations/laboratory tests.
- Anti-venom treatment.
- Observing the response to anti-venom.
- Deciding whether further dose(s) of anti-venom are needed.
- Supportive, ancillary treatment.
- Treatment of the bitten part.
- Rehabilitation.
- Treatment of chronic complications.

Diagnosis of poisoning [3, 21].

The diagnosis in a case of poisoning can be made from the

1) History
2) Physical Examination
3) Laboratory Evaluation
4) Toxicological Screening

1. History:

- Most important indicator of toxic ingestion. Careful history regarding involved toxins, amount of drug and timing should be recorded.
- Information regarding prescription medication, over the counter drugs and illicit substances of abuse should be obtained.

- Friends, relatives and other involved health care providers should be questioned and medications identified.
- Medication found on or near the patient should be examined and pharmacy on the medication label should be called to determine the status of all prescription medication.

2. Physical Examination

- Evaluation of airway patency, Respiration, Circulation.
- Rapid assessment of mental status, temperature, pupil size, muscle tone, Reflexes, skin and peristaltic activity.
- Separate patients into two groups

1) Depressed Status

2) Agitated Status

Drugs causing the **depressed** status are Sympatholytic like adrenergic blockers, anti-arrhythmias, anti-hypertensive, anti-psychotics, cyclic anti-depressant, Cholinergic like nicotine, carbamates, organophosphates, physostigmine, pilocarpine, Sedative-hypnotics like alcohols, barbiturates, benzodiazepines. Narcotics like analgesics, anti-diarrheal agents; and others like CO, cyanide, hypoglycemic agents, lithium and salicylates.

Drugs causing an **agitated status** are sympathomimetic like adrenergic agonists, amphetamines, caffeine, cocaine, ergot alkaloids, MAO inhibitors, theophylline, anti-cholinergic like anti-histamines, anti-parkinsonism drugs, anti-psychotics, anti-spasmodic, cyclicantide present, cyclobenzaprine; drug withdrawal like a blockers, clonidine, ethanol, opioids, sedatives-hypnotics, hallucinogens like LSD, marijuana.

• Pupil size and reaction to light, with the patient's physiological status gives a rapid clue regarding dominant ingestion.

- Pinpoint pupils with - Phencyclidine agitation intoxication
- Pinpoint pupils with Lethargy -Narcotic overdose
- Dilated pupils reacting to light -Cocaine intoxication
- Dilated pupils not reacting to light-Anti-cholinergic intoxication

- **Laboratory evaluation [21].**

Clinical laboratory data include assessment of the three gaps of toxicology

1. The Anion gap
2. The osmolal gap
3. The arterial oxygen saturation gap.

Unexplained widening of the difference between calculated and measured determination of these values raises the suspicion of toxic ingestion.

1. Anion Gap: refers to the difference between measured Cations and measure Anions.

$$AG = [Na^+] - [Cl^-] - [HCO3^-]$$

Normal Value = 12 ± 4 $^{meqL-}$.

The presence of anion gap indicates that there are more unmeasured anions than cations, since total serum cations equals total serum anions. Unmeasured cations include K^+, Mg^{++} and Ca^{++} totaling about 11 $^{meqL-}$under normal conditions, and the concentration of unmeasured anions including protein (mainly albumin), sulfates, phosphates and organic acids is about 23^{meqL-1}.

The anion gap falls by 2.3 $^{meqL-1}$ for every 1 gmL-1 decrease in plasma albumin concentration.

2. Osmolal gap: (measured – calculated osmolality).

Certain drugs and toxins of low molecular weight produce a discrepancy between measured and calculated plasma osmolality, commonly referred to as the osmolal gap. (osmolal gap equals measured minus calculated osmolarity).

Normal Plasma Osmolality=285–295 mosmlL-1and is calculated as:

Calculated osmolality = $2[Na^+] + [BUN]/2.8 + [Glucose]/18 + [ethanol]/4.6$

Where Na+(in mmolL-1) is multiplied by 2 to account for anions(Cl- and HCO-and BUN and glucose are divided by 2.8 and 18 to convertmgdL-1 to mmolL-1 and ethanol is divided by 4.6.

3. Oxygen saturation gap:

Toxins associated with an elevated arterial oxygen saturation gap [>5% difference between saturation calculated from ABG determination and saturation measured by co-oximetery] including carbon monoxide and methemoglobin.

These toxins interfere with oxygen binding to haemo-globin and thereby significantly decrease oxygen content without lowering PaO_2. It is important to note that Oxygen saturation measured by pulse oximetry is also falsely high in the setting of these toxins.

H_2S and cyanide interfere with cellular utilization of oxygen leading to abnormally high venous oxygen saturation or "arterializations of venous blood".

Obtain serum electrolytes, BUN, Blood glucose and serum osmolality.

• Calculate anion and osmolal gaps.

• Obtain ECG; look for widened QRS, and QT interval sand AV block.

• Obtain CXR to look for pulmonary edema or inflates.

• Obtain Abdominal X-Ray to look for radio opaque pills.

• Obtain urine for toxicological screening and routine analysis. Look for calcium oxalate crystals.

• Pregnancy test in women of child bearing age.

4. Toxicological Screening.

It provides direct evidence of ingestions, but it rarely impacts initial management and initial supportive measures should never await results of such analysis.

It is used to;

- Provides ground for treatment with specific anti-dote or method for enhancing drug elimination.

- Also identifies drugs that should be quantified to guide subsequent management. Also look for characteristic signs of various kinds of poisoning while immediate treatment measures are being started.

Management of Poisoning [22, 23, 24].

Treatment goals include support of vital signs, prevention of further poison absorption, enhancement of poison elimination administration of specific antidotes and prevention of re-exposure. Majority of poisoned patient require only supportive treatment.

Initial Therapy

Immediate management of life threatening conditions in victims of poisoning with coma, seizures or marked airway obstruction should be as follows.

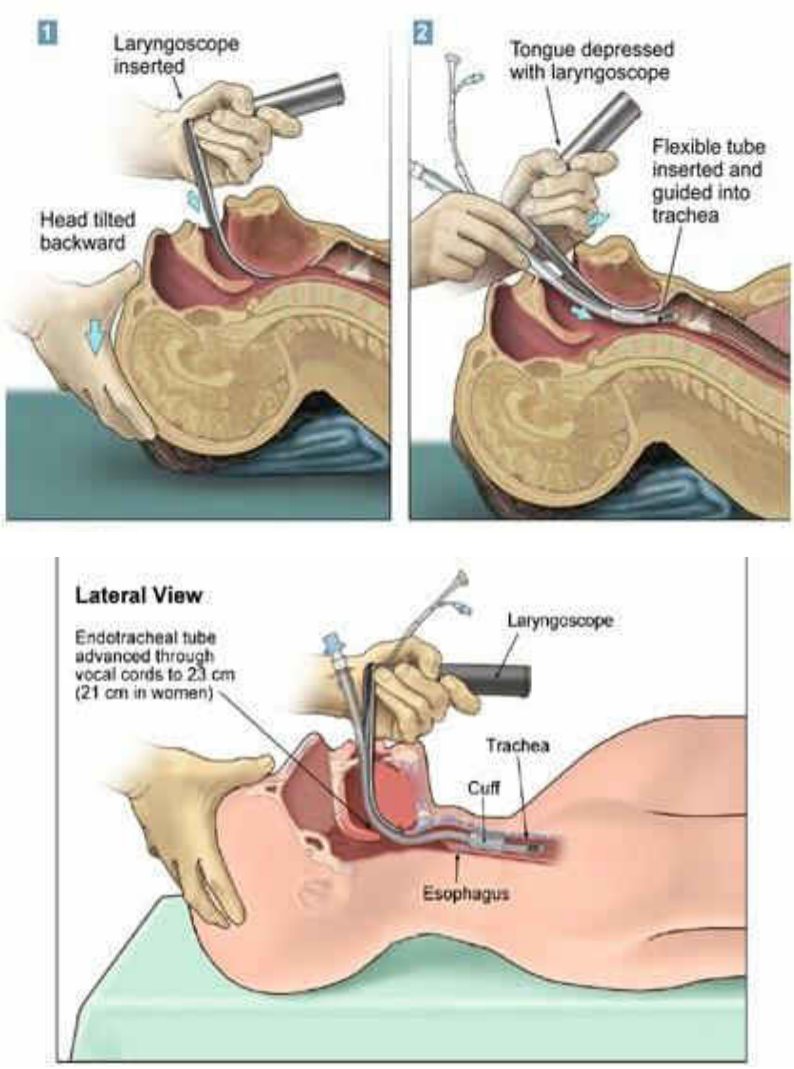
A. **Keep Airway open**: Establish and maintain an adequate airway and ventilation.

• Begin supplemental oxygen 5-10, it's by nasal prongs or mask.
• If the patient has no gag reflex, intubate the trachea with a cuffed endotracheal tube as soon as possible to:
a) Protect the airway
b) Facilitate oxygenation and ventilation
c) Helps removal of secretions.

• Indications for endo-tracheal intubation
1. Patients in coma or with markedly depressed gag reflex.
2. Awake patient with normal gag reflex.
3. Lethargic patient with fluctuating mental status and a variable gag reflex.

Choice of Intubation technique.
a) Oro-tracheal intubation.
b) Naso-tracheal intubation.

- **Orotracheal intubation** - This technique is useful for the comatose patients, since it is rapid and the location of E.T. tube can be verified by direct vision.



→Orotracheal intubation.

Figure 20: Orotracheal intubation.

- **Nasotracheal intubation**. – This technique is useful in the agitated patient. It doesn't require jaw relaxation and has the advantage of not requiring neck manipulation in a patient who may have cervical spine injury.

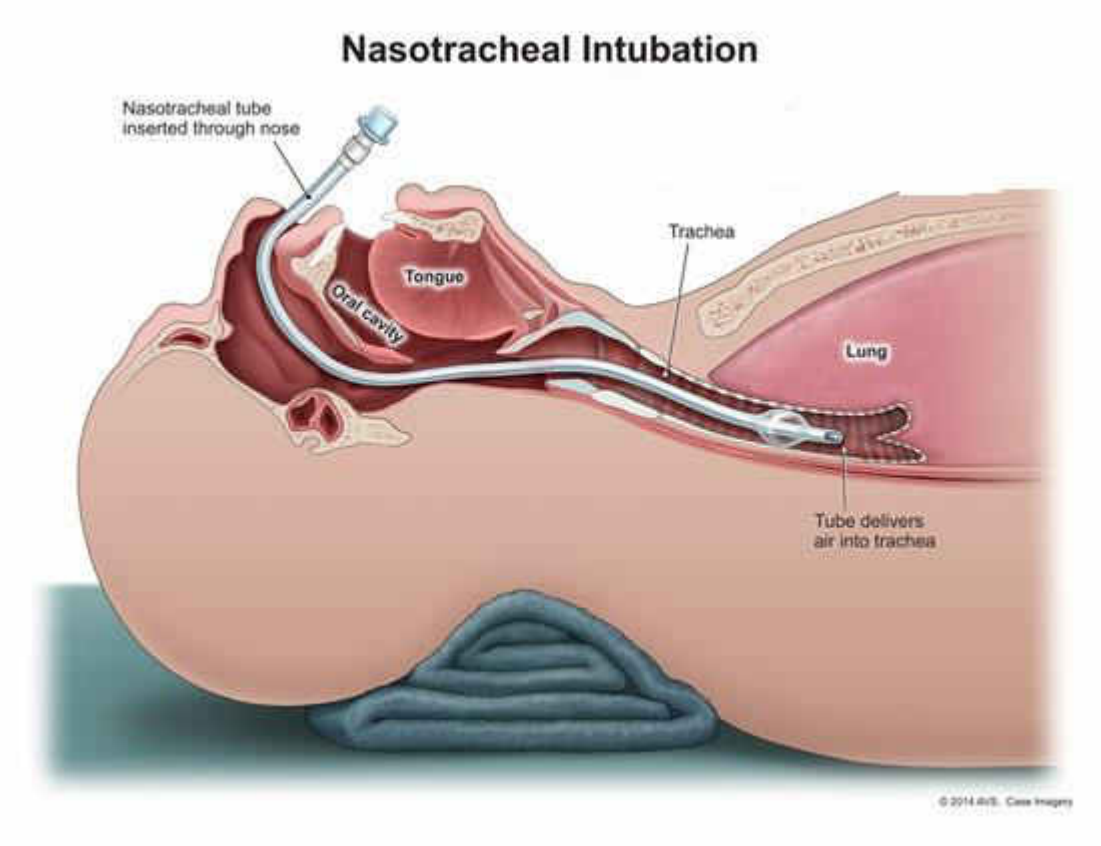


Figure 21: Nasotracheal incubation

B. **Obtain Arterial Blood Gas measurements:** to determine adequacy of ventilation and perfusion.

C. Gain Intravenous Access: Insert an 18G peripheral or central line and draw blood for complete blood count, serum electrolyte and blood glucose measurements and tests of hepatic and renal function.

D. **Treat Coma** (COMA COCKTAIL)

1. Give glucose, 50ml of a 50% solution (25g glucose) IV over 3-4 minutes.

2. Give Naloxone, 0.4 – 2 mg I.V. If patient's response is weak or if narcotic overdose is suspected, give repeated doses of 2 mg every 1-2 min up to a total of 10-20 mg. Patients responding to naloxone must be observed for at least 3 hours after the last dose of naloxone.

3. If Alcoholism or Malnutrition is suspected, give thiamine, 100mg I.V/I.M. The level of consciousness of all intoxicated patients should be assessed and the time of assessment should be recorded.

4. Give Flumazenil, 0.2-0.5 mg. I.V. repeated every 30 sec. up to 3 mg. in case of suspected benzodiazepine poisoning.

E. Maintain Circulation: Restore the intravascular volume by intravenous infusion of crystalloids. If the administration of more than 20-30 mlKg-1 of crystalloid solution and usual doses of dopamine (5-15 mgkg-1min-1) fail to restore blood pressure, insert a pulmonary artery catheter to obtain pressure reading and help guide further with fluids and pressure agents.

F. **Treat Seizures**: Give diazepam 0.1 to 0.2 mg/Kg IV over 1-2 minutes. Followed by Phenobarbital 15 mg/kg IV if there is no response to diazepam.

G. **Start ECG monitoring**-Obtain 12 lead ECG note the rate of rhythm, presence of arrhythmias and PR, QRS and QT intervals.

H. Perform Gastric cleavage: Collect sample for future toxicological analysis if required.

I. Search for associated Illness:

Look for cause of coma or seizures

Look for

a) Head trauma
b) Hemorrhage or shock
c) Infection
d) Metabolic disorders
e) Hypothermia
f) Hyperthermia

J. Document: The available history and the level of coma before ambulance personnel, relatives etc. leave the hospital.

FUNDAMENTALS OF POISONING MANAGEMENT [25, 26, 27, 28, 29, 30,].

1. SUPPORTIVE CARE

• Airway protection.
• Treatment of arrhythmia.
• Oxygenation / Ventilation·
• Hemodynamic support.
• Treatment of arrhythmia.
• Correction of metabolic derangements.
• Prevention of secondary complications.

2. PREVENTION OF FURTHER POISON ABSORPTION

• Gastrointestinal decontamination.
• Decontamination of other sites.
• Induced emesis, Gastric cleavage.
• Eye decontamination.
• Activated charcoal.
• Skin decontamination.
• Whole bowel irrigation.
• Body cavity decontamination.
• Catharsis.
• Dilution.
• Endoscopic/surgical removal.

3. ENHANCEMENT OF POISON ELIMINATION

• Multi dose activated charcoal.
• Extracorporeal removal.
• Forced diuresis.
• Peritoneal dialysis.
• Alteration of pH

• Hemodialysis.

• Chelation.

•Hemo-perfusion /hemofiltration.

• Exchange transfusion.

• Hyperbaric oxygenation.

4. ADMINISTRATION OF ANTI-DOTES

• Neutralization by antibodies

• Metabolic antagonism

• Neutralization by chemical binding

• Physiologic antagonism

5. PREVENTION OF RE-EXPOSURE

• Adult education.

• Notification of regulatory agencies.

• Child proofing.

• Psychiatric referral.

PREVENTION OF FURTHER DRUG ABSORPTION

Inhaled Poisons

• Remove the patient from the source of the poison. Give oxygen by mask (CN poisoning).

• Inhalation of water aerosol may dilute inhaled irritant in the naso-pharynx.

• Check for hoarseness and singed nasal hairs.

• Be alert for delayed development of upper airway obstruction and pulmonary edema.

Contaminated Eyes

• Wash the Eyes with the copious amounts of plain water or (hang a bottle of 500-1000 ml of NS) above the patient and dribble the solution slowly into the corner of the eye through the I.V tubing.

• Check the tears with pH paper after the eyes have been washed to make sure that all toxic material has been removed.

• A careful eye examination is done following irrigation.

Contaminated skin

• Wash the skin immediately with plenty of water and dilute soap solution.

• Discard contaminated clothes in a marked plastic bag. Organophosphate compounds are well absorbed through the skin and are difficult to remove.

Ingested Poisons

a. Emesis is recommended for emergency treatment of drugs not adsorbed by activated charcoal. It is induced with syrup of Ipecac. Dose is 15ml orally for children (5-10 ml for 6m-1yr) and 30ml for adults. Give2-3 glasses of plain water following this. This induces the vomiting within 20-30 min. and can be repeated after 30 min. The vomits should be inspected for remnants of pills or toxic substances, its appearance and odor should be noted. Apo-morphine is a parenteral emetic, and can be used with caution because of its CNS and cardiac side effects. Do not induce emesis in a comatose patient.

Contraindications to the induction of emesis are

- Caustic (alkali) or corrosive (acid) ingestion.
- Agents that rapidly produce coma or convulsions in less than 30 min. and may predispose to aspiration during emesis.
- Prior significant vomiting.
- In infants less than 6 months of age.
- In foreign bodies.
- Absence of bowel sounds.

b. Gastric cleavage: It is done after suspected serious ingestion when attempt to induce emesis fail and when patients are uncooperative or lethargic, or when gag reflex is markedly depressed. Place the patient in left lateral decubitus position with head down. It is performed with a large bore nasogastric tube. Use tap water or saline at body temperature in 250ml increments and continue cleavage until fluid returns clear. Airway must be protected if gag reflex is depressed.

c. Activated Charcoal: It has powerful adsorption capacity and is given orally or via gastric tube. It irreversibly binds the drugs within the bowel and reduces the blood concentration by reducing drug absorption and by creating a negative diffusion gradient between the gut lumen and blood (gastrointestinal dialysis).The dose of activated charcoal is 1gmKg-1 orally with a minimum of 15 gm. The usual dose is 60-70 gm. It can be mixed with 4 parts of water and given every 4 hourly as long the bowel sounds are present. It is used for most poisons accept alcohol, potassium, Fe and Li.

Contraindication to the use of activated charcoal

a) It should not be given before, concomitantly or just after ipecac because it may absorb the ipecac and interfere with its emetic properties.

b) It should not be given before, concomitantly or just after oral anti-dotes unless proved not to interfere significantly with their absorption.

c) It does not effectively adsorb caustic and corrosive and may produce vomiting or stick to the mucosa of oesophagus or stomach and may look like burn on endoscopy.

d) It should not be given if no bowel sounds are present.

e) Activated charcoal is a stool marker, indicating that the toxin has passed through the GIT and no further significant absorption from the original ingestion will occur.

d.Catharsis: Cathartic salts (disodium phosphate, magnesium citrate and sulphate, sodium sulphate) or saccharide (mannitol, sorbitol), promote the rectal evacuation of gastrointestinal contents. The dose of sorbitol is 1-2 gkg-1.Their aim is to prevent constipation following charcoal administration. They are contraindicated in corrosive poisonings and pre-existing diarrhea.

e. Whole bowel irrigation: It is performed by administering a bowel cleansing solution containing electrolytes and polyethylene glycol orally or by gastric tube at a rate of 0.5 Lhr-1 in children and 2 Lhr-1 in adults until rectal effluent is clear. The patient must be in sitting position. It is useful in patients who have ingested foreign bodies, packets of illicit drugs, slow releasing or enteric coated medicines or heavy metals. It is contraindicated in patients with bowel obstruction, ileus, hemodynamic instability, and compromised unprotected airway.

Enhancement of the absorbed drug

The decision to use measures to enhance drug elimination should be based on a rational understanding of drug properties and the clinical condition of the patient.

The various methods are:

- ✓ **Diuresis and pH manipulations:** Since many toxins are weak acids or bases, they can be ionized in solutions of varying P^H. In the Ionized state, they are less likely to cross cell membranes and their re-absorption by renal tubular epithelium is decreased.

(a) Weak acids such as salicylate and phenobarbital are more fully ionized in basic solutions, so that alkalinizing the urine may serve to trap them in the tubular lumen, thus increasing excretion of the drug in the urine

.

(b) Weak bases such as amphetamines, strychnine and phencyclidine are more ionized in acid medium; acidification of urine has been proposed to enhance their removal. Contraindication to forced diuresis includes severe CHF, cerebral failure and pulmonary edema.

1) Extra Corporeal Removal of Toxins:

(a) **Hemodialysis**: During hemodialysis, toxin is removed from the blood into a dialysate solution across a semipermeable membrane. The toxin must be relatively water soluble and not highly protein bound. It should have a small volume of distribution and slow rate of intrinsic elimination (a long t ½). It is effective in removing methanol, ethylene glycol, salicylates and lithium. It is also of value in correcting pH and electrolyte imbalances especially in an uricpatients.

Criteria for potential dialyzalibility include
a)Water solubility
b) Low molecular weight
c) Protein binding
d) Volume of distribution and
e) Intrinsic clearance of the substance.
Complications of hemodialysis include intravenous access complications, hypophosphatemia, alkalemia, disequilibrium syndrome and hypotension.

b) **Peritoneal Dialysis**: Peritoneal Dialysis is only 1/8-1/4 as efficient as hemodialysis and is not a preferred method.

c) **Hemoperfusion:** is defined as direct contact of blood with a sorbent system. In hemo-perfusion, blood is pumped through a column of adsorbent material (Charcoal or resin) and returned to the patient's circulation. Vascular access similar to that for hemo-dialysis is required. The kinetic conditions are the same as in hemo-dialysis. It is commonly associated with thrombocytopenia. It will not correct P^H or electrolyte imbalance.

d) **Hemofiltration:** Hemofiltration is potentially useful method for removal of substances with a large V_d slow inter compartmental transfer and vivid tissue transfer. Arteriovenous (CAVH) or venovenous (CVVH) methods are used.

e) **Antidotes:** An antidote is any substance that increases the mean lethal dose of a toxin, or that can favorably affect the toxic effects of a poison.

Table 4: List of poisonous drug and their anti-biotic's.

POISONS	ANTIDOTES	DOSAGE REGIMEN
Anti -chollinergic agents	Physostigmine	2 mg iv over 5 minutes, continue agents with an infusion of 4-6 mg hourly (adult dose)
Anti-cholinesterase	Atropine	1-2 mg i.v. repeated 2-4mg every 5-10 min. or atropine drip.
Anti-coagulants (warfarin type)	Vitamin K	2-5 mg iv adult, 0.4 mg/kg child
Organophosphates	Atropine Pralidoxime	2 mg iv (IM or SC in less severely poisoned patients) followed by further two doses at 5-10 minutes interval until full atropinisation.
Benzodiazepines	Flumazenil	Initially 0.2 mg IV over 30 seconds. Further doses of 0.5 mg can be given over 30 seconds at 60 seconds interval to a total dose of 3 mg.
Narcotic analgesics	Naloxone	0.8-1.2 mg i.v. (children 0.2 mg) Repeat if respiratory depression not reversed within 1-2 mins.
Carbon monoxide	Oxygen (normobaric hyperbaric)	Administer as high as inspired or oxygen as possible until carboxyhaemoglobin concentration falls below 5% - hyperbaric oxygen in severe cases.

Methaemoglobin	Methylene blue	0.2 $mlkg^{-1}$ of 1% solution. Slowly I.V. over 5 minutes. Repeated as necessary upto 6 mgkg-1.
Acetaminophen	n-acetylcysteine	140 $mgKg^{-1}$ orally followed by 70 mgKg-1 every 4 hourly for 17 doses or 6 doses if no Hepatotoxicity.
Paracetamol	Acetylcysteine	300 $mgkg^{-1}$ over 16 hours
	Methionine	2.5 gm orally every 4 hours for 12 hours.
Cyanide	Dicobalt edentate Sodium nitrite Sodium Thiosulphate Oxygen	300 mg iv over 3 minutes 10 ml of 30% iv over10 minutes. 50 ml of 25% solution in over 10 minutesin may be upto 4 gm I.V. Administer inspired oxygentill clinical recovery occurs.
Thallium	Berlin blue	250 $mgkg^{-1}$ per day in divided doses till thallium level is < 10mgL-1 in blood and urine.
Ethylene Glycol	Ethanol	Dose given should be sufficient to maintain plasma ethanol levels at 1-2 gL^{-1}.
Methanol	Ethanol	Dose given should be sufficient to maintain plasma ethanol levels at 1-2 gL^{-1}.

β-Blockers	Glucagon Isophrenaline	5 mg iv over 1 minute followed by an infusion of 1-10 mgh^{-1},10-50 mgmin-1 I.V
Digoxin Digitoxin	Fab antibody fragments	Dose should match estimated Digitoxin .
Ca- channel Blockers	$CaCl_2$, Cagluconate	10 ml of 10% $CaCl_2$ or30 ml of 10% Cagluconate over 2 min.0.2 $mlKg^{-1}hr^{-1}$10mlKg-$1hr^{-1}$.
Heavy metals (lead, mercury, arsenic)	DMSA(Dimercapto succinic acid)	30 mgkg-1 8 hourly for 5 days (then 20 mgkg-1 12 hourly for 14 days
	DMPS(Sodium 2,3 Dimercaptopropane Sulphonate)	Chronic: 100 mg 3 times a day. Acute: 250 mg every 4 hours for 24 hours then 250 mg every6 hours for the next 24 hours
	Sodium calcium edenate	Upto 40 $mgkg^{-1}$ twice daily by iv infusion repeated every 48 hours until level falls below toxic levels.
	Dimercaprol	Mercury: 2.5-3 $mgkg^{-1}$ deep IM injection 4-hourly for 2 days, 2-4 times on third day. .
	Pencilamine	Lead: 0.5-1.5 g per day orally for 1-2 months or until lead levels falls below toxic level.

Iron salts.	Desferrioxamine	In severe iron poisoning (> 90 mmolL-1) upto 15 $mgkg^{-1}$ per hour reduced to keep the total iv dose under 80 $mgkg^{-1}$in each24 hours.
Coral snake Rattle snake bite	Anti-venom	Loading dose 1mgKg $^{-1}$upto 100 mg. Anti-venom is diluted in 1000 ml of saline for adults or 20 ml for child

Supportive care:-

The goal of Supportive therapy is to maintain physiologic homeostasis until detoxification is accomplished and to prevent and treat secondary complications.

Respiratory Complications:-

Are the commonest causes of death after acute poisoning and immediate management must be given priority to airway ventilation and prevention of aspiration, Hypoventilation, hypoxia and pulmonary edema should be treated. Treatment consists of O_2, fluid restriction diuretics, mechanical ventilation with PEEP, dialysis.

Cardiovascular complications:-

Include hypotension, cardiac arrhythmias and cardiac arrest. Treatment should be on the lines of peripheral perfusion and maintenance of myocardial function.

Renal Failure:–

May be due to tubular necrosis because of hypotension hypoxia or direct effect of poison on tubular cells. Haemoglobinuria or myoglobinuria may further precipitate renal failure. Patient should be catheterized to maintain a urine output of 0.5 mlkg-1hr $^{-1}$ with volume resuscitation or dopamine infusion (2-4gmkg $^{-1}$min-1).

Neurological complications:–

Include depression of consciousness level, seizures, cerebral edema and peripheral nerve injuries as a result of prolonged pressure. Therapy consists of correction of ABG and metabolic abnormalities and hypotension, reduction in intracranial pressure, hyperventilation,

elevation of head and fluid restriction. Seizures may be because of metabolic disturbances and cerebral hypoxia and direct toxic effect. Treatment by I.V. diazepam phenobarbitone or infusion of thiopentone.

Hypothermia:–

Should be treated by passive re-warming, I.V. fluid warming, and warm water humidifierin artificial ventilation. Temperature should be monitored centrally via esophagus or naso-pharynx and ECG should be monitored for arrhythmias.

Hyperthermia:–

May occur with tricycle anti-depressant cocaine or amphetamine etc. Active cooling with sedation, paralysis and ventilation may be required to control core temperature.

Metabolic complications:–

The concentration of urea and electrolytes and blood glucose, ABG should be checked routinely. Sodium bicarbonate may be needed if pH falls below 7.1.

Hematemesis:–

Can be produced by caustics, corrosives, iron, mercury, arsenic, lithium, phosphorous, fluoride mushroom, plant poison and organophosphates. The therapy consists of iced saline Cleavage, fluid and blood replacement ant-acids H_2 blockers may be used.

Hepatic and gastrointestinal complications:–

Gastric stasis may occur in comatose patients or in anti-cholinergic or opioid poisoned patient. Early decompression with ryles tube and stress ulcer prophylaxis may be used.

LITERATURE REVIEW

***JAISWAL A.K* (2014):** Studied about the quantitative estimation of Zinc using trace metal analyzer in Zinc Phosphide poisoning cases. Zinc phosphide which is readily available rodenticide in India. Silver nitrate test is performed to analyze the Phosphide poisoning [31].

***TSAKALOF* (2007):** Studied the analytical methods of biological monitoring for exposure to pesticides. Extensive use of synthetic pesticides for agricultural & non- agricultural purpose began in last 50 years. So their wide & extensive application exposure to hazardous pesticides is a concern to the general population & occupationally exposed persons. **Robust Methods** are needed for measuring markers of pesticide exposure. This study mainly conducted by quantification of pesticides metabolites & parent compounds in different biological samples. These studies require development of sensitive, selective, reliable analytical methods with high sample throughout and at the same time, acceptable costs. Most of the methods used in now a days have LODs in the low- mg/ml range or even lower, and this enables monitoring of occupational & occasional environmental human exposure. LC-MS/(MS),GC-MS& GC- MS/MS are most commonly used in the detection. In some class of pesticides, and especially for organo-chlorine pesticides the GC-ECD is used [32].

***BRADY W.J* (2001):** Analyzed the electro cardio graphic manifestations in digitalis toxicity. This study mainly based on the Electro cardio graphic findings. ECG manifestations of therapeutic digoxin- "Probable digitalis effect''- four findings on ECG that are consistent with the presence of digitalis. The earliest finding is that of T-wave changes virtually any form. The second finding is Q-T interval shortening from a decrease in ventricular repolarization time. Third finding is the classic sagging or scooped appearance of the ST-segment with concomitant ST-segment depression. This finding is more pronounced in leads with tall R-waves such as the lateral leads. Lastly one may find an increase in the U-wave amplitude. Ectopyis common manifestation of digitalis toxicity. Premature ventricular beats (PVCs) both univocal& multi focal are often earliest dysrhythmia associated with digitalis intoxication. Digoxin continues to be a common medication utilized by patients presenting to the ED, the non-specific sign& symptoms make the diagnosis of toxicity difficult to establish, the key to successful management is early diagnosis. No single dysrhythmia is always found, PAT-with block, Junction tachycardia, and bidirectional ventricular tachycardia are common. The digitalis toxicity is increased automaticity with concomitant conduction delay, Digoxin

specific Fab fragments have revolutionized the treatment of digitalis toxicity and should be considered early in the severely poisoned patient [33].

***WAHABA* (2008):** Studied about the acute aluminum phosphide poisoning. Acutealuminum phosphide poisoning is an extremely lethal poisoning. Ingestion is usually suicidal in intent, uncommonly accidental, and rarely homicidal. This study mainly focused the mechanism & silent features of Aluminum Phosphide poisoning along with the diagnosis & management strategies. A positive history of ingestion is the basis of diagnosis in most cases. The presence of typical clinical features, garlicky dour from the mouth & highly variable arrhythmias in a young patient with shock and no previous history of cardiac disease points towards aluminum phosphide poisoning. Confirmation can be done by silver nitrate test [34].

In this test 5ml of gastric aspirate and 15ml of water are put in a flask is covered by filter paper impregnated with 0.1N silver nitrate. The flask is heated at 50° c for 15-20 minutes. If phosphine is Present the filter paper turns black. For performing test on exhaled air, the silver nitrate impregnated filter paper is placed on the mouth of the patient and the patient is asked to breathe through it for 15-20 minutes, blackening of the paper indicates the presence of phosphine in Breath. Most specific & sensitive method for detecting the presence of Ph 3 in blood air in gas chromatography [34].

***STEPHEN R.A* (2012):** Analyzed the diagnostic method of Carbon monoxide poisoning by "Novel magnetic resonance imaging pattern.(MRI). Carbon monoxide poisoning (CO) is common, potentially fatal& probably under diagnosed because of its non-specific clinical presentation. The addition of MR- imaging to the diagnostic workup of co-intoxicated patients offers additional information to clinicians to better gauge patient outcome, especially when other parameters are equivocal. Outpatient follow up is required to assess for the development of *delayed neuropsychiatric Syndrome* (DMS) which can be cause personality changes, memory difficulties, and gait disturbance. Carbon monoxide poisoning should be included in the differential diagnosis in patients found to have cerebellar white matter lesions on imaging [35].

***ZHANG H* (2014):** Performed the treatment of acute thallium poisoning. The strategies for the treatment of acute thallium poisoning include oral administration of Prussian blue, diuretic, laxative and potassium supplement. Prussian blue can bind to thallium, together with catharsis, promoting the gastrointestinal excretion of thallium through the stools. Supplement

of potassium is used to increase the concentrations of plasma potassium, which can enhance ion exchange between potassium ions and intracellular thallium, thereby enhancing urinary excretion of thallium via the kidneys. They found that after treatment with potassium, together with other specific therapies, the concentrations of blood and urinary thallium were rapidly reduced. They believe that potassium treatment may contribute to the clearance of thallium in those patients. They found that blood purification therapies, together with traditional Prussian blue, were effective in the clearance of thallium in the body [36].

***HONG-X L* (2015):** Analysed the clinical study of continuous micro-pump infusion of atropine and pralidoxime chloride for treatment of severe acute organo-phosphorus insecticide poisoning. They study sought to assess the effectiveness of a constant micro-pump infusion of atropine and pralidoxime chloride compared with repeated-bolus doses in patients with severe acute organo-phosphorus insecticide poisoning (aopp). The others research study found that continuous micro-pump of a combined atropine and pralidoxime chloride infusion was a greater benefit to patients with severe aopp than the intermittent injections of atropine and pralidoxime chloride given to the control group. During the course of treatment, the time to atropinization and time to ache recovery was shorter in the experimental group than the control group, leading to a lower amount of intravenous atropine needed for atropinization given to the experimental group than to the control group. The apache ii, (acute physiology and chronic health evaluation ii) score at atropinisation was also significantly better in the experimental group than in the control group. Compared with that in the control group, there was a lower case fatality rate (10% vs. 26.7%) in patients given a micropump infusion of atropine and pralidoxime chloride [37].

***ZUBIAUR O* (2015):** Performed the therapeutic psychotropic drugs: most common cause of unintentional poisoning in children the aim of them study to determine the most common substances involved in unintentional poisoning in children attending paediatric emergency departments (ped) in Spain. They make a descriptive study was conducted based on a prospective registry of the poisonings registered in the 57 ped participating in the toxicology surveillance system of the Spanish society of paediatricemergencies. They take a total of 639 poisoning were registered during the study period, 459 of them (71.8%) were unintentional. The most commonly involved substances were drugs (253, 55.1%) followed by household products (137, 29.8%). The drug groups most involved were psychotropic drugs (62, 24.5%), which included benzodiazepines (54), anti-catarrhal (41, 16.2%), and antipyretics (39,

15.4%). And finally the authors concluded that the most common reason for consulting spanishpeds is the unintentional ingestion of psychotropic drugs, mainly benzodiazepines [38].

***MANGARAJM M* (2014):** Concluded the abnormalities in patients with Organo phosphorous poisoning. The study depicted the signs and symptoms in the study group. The others concluded that the op compounds are quickly absorbed through the respiratory system, gastrointestinal tract mucosa as they are lipophilic and act by inhibiting the enzyme acetyl cholinesterase leading to excessive accumulation of acetylcholine at the synapses and myoneural junction. This causes overstimulation of muscarinic and nicotinic receptors at the synapses within the central and peripheral nervous system, producing an array of symptoms like miosis, bradycardia, increased gastrointestinal motility, emesis, sweating, tachypnoea, salivation, lacrimation, altered sensorium, fasciculation, bronchospasm, blurred vision, urination and defecation. The complications include acidosis, respiratory paralysis, acute renal failure, seizures, arrhythmia, aspiration etc and death may be due to combination of one or above complications the most common nicotinic and muscarinic manifestations like miosis (78%) vomiting and nausea (50%), sweating (48%) were observed in the study group. Laboratory parameters like plasma glucose, serum urea, and creatinine and liver enzymes showed a significant rise from grade 1 to grade 3, reflecting organ damage due to toxicity of poisoning [39].

***SHILPA N.D*(2014):**The author described the study of acetyl cholinesterase, butyrylcholinesterase and β - glucuronidase in organophosphorus poisoning .finally they results indicated that activities of acetyl-cholinesterase and butyryl-cholinestrase decrease in mild, moderate and severe organophosphorus poisoning in proportionate manner whereas, β-glucuronidase activity increases as severity of organophosphoruspoisoning progresses. Thus, all the three enzymes showed alterations in their activities however, the degree of change in activity was maximum in case of acetylcholinesterase. Thus acetylcholinesterase activity is the most sensitive marker amongst three enzymes in organophosphorus poisoning [40].

***PANDA S* (2007):** Analysed laboratory abnormalities in patients with organophosphorous poisoning. Organophosphorous (op) poisoning is an ever increasing and troublesome situation in the developing countries and is a major health care challenge in the 21st century. Hundred patients who attempted suicides with organophosphates, admitted to the emergency services were included in there study. They were graded (grade 0,1,2,3) according to clinical findings and examined for parameters like rbs, serum urea, serum creatinine, liver function

tests, serum amylase, serum cholinesterase and ldh . The apache (ii) of the cases were determined and correlated with severity of the clinical manifestations. A significant decline in serum cholinesterase (che) with increasing grades of intoxication ($p < 0.05$) was observed along with raised levels of random blood sugar, serum urea, creatinine, hepatic enzymes and amylase. Apache (ii) score, showed a significant rise with severity of the degree of intoxication ($p<0.001$) and a negative correlation with serum cholinesterase. They findings of this study highlighted usefulness of biochemical and clinical indices in the management of organophosphorous poisoning thereby recognizing the complications early and facilitating early management [41].

***SUPREETI B* (2013):** Studied about the rationally used antidotes in organophosphorus poisoning prevent suicidal death. This retrospective study was undertaken in a block primary health centre to evaluate the utilization of two antidotes, atropine and pralidoxime, in organophosphorus poisoning (opp). This study was carried out with data from prescription record sheets of opp patients. Data was statistically analyzed in respect of demographic profile, signs of atropinization and the health outcome after treatment with antidotes. Demographic profile of obtained 181 opp cases revealed 100% suicidal tendency, 72.93% female, 70.17% in age group of 15-24 years and 84.53% from rural population. Nausea, vomiting, excessive salivation, sweating, meiosis (82.20%), blurred vision and disturbances of consciousness (7.93%) were the main presenting sign-symptoms. Following stomach wash, 17.12% patients improved; rest 82.88% received antidotes (atropine to all and pralidoxime to 58.66% cases). Them concluded the profile on opp consumption and treatment reveals all patients got stomach wash with normal saline; of which 17.12 % with time lag of <1 hour got cured and discharged following an observatory period, rest 150 patients received specific antidotes with titrated doses of atropine and/or pralidoxime. About 41.33 % patients were cured with atropine alone after stomach wash with time lag of 1-3 hours. Whereas, 34.66 % patients needed pralidoxime in addition to be cured with a time lag of 3-5 hours. About 24 % patients were referred to the higher centre even after proper treatment. Among the categorical responses after atropinization) 83.33% cases showed positive responses. Few patients required other supportive medications like theophylline (18 %), dexamethasone (12 %) and diazepam (10 %) [42].

***CHANDRA SW* (2014):** Studied about the investigational profile of common pesticide poisoning cases admitted at saims, indore. A prospective study was done in shrieurobond medical college hospital, indore during 1st January 2012 to 20th august 2013. 110 cases were admitted with history of pesticide poisoning during this period, out of them 10 cases were excluded, as they left the hospital within 1-2 hours of hospitalization. Most common poison was organophosphorus (46 cases). Rodenticides 14 cases, aluminium phosphide 12 cases, ethylene di bromide 5 cases, others 3 cases and unknown poison was consumed by 2 peoples. Investigations like liver function test, renal function test and electrolytes were studied in common pesticide poison i.e. Organophosphorus, aluminium phosphide and ethylene di bromide (edb). Liver function test was raised in most of the cases of ethylene di bromide poisoning. Renal function test (rft) was normal in most of the cases of organophosphorus and aluminium phosphide and edb poisoning. Sodium, potassium and chloride showed normal levels in most of the poisoning. Hence forth this study was undertaken to facilitate the clinicians and toxicologist for better judgment in regards to patient condition and co-relation with various investigative findings. Due to the risk involved in treatments of pesticide poisoning, there is general agreement that emphasis should be on preventing pesticide illness rather than relying on treatment. SGOT was raised in most of the cases of edb poisoning. Rft and electrolytes showed normal results in most of the cases of above mentioned three pesticide poisoning [43].

***SURENDRA K* (2016):** Analysed prospective study of current trends of poisoning: an experience at a tertiary care hospital hadoti region, rajasthan, India: the study was done in patients admitted with history of poisoning in the department of medicine at m.b.s hospital, kota;rajasthan, India from the year july‘2009 to December 2011 this study includes 799 consecutive poisoning patient who were admitted to medicine department. Detailed history and clinical examination were done in all patients. During the study period 55,428 emergency cases were admitted, out of which 799 cases were of poisoning (1.41%). Out of 799 cases there were 674 cases of suicidal poisoning and 125 cases of accidental poisoning. The highest number of poisoning cases were in lower socio-economic status 592 cases (74.09%), followed by middle class 171 cases (21.4%) and then the upper class which constituted 47 cases (5.88%). Insecticides group which constituted 245 (30.65%) which was most common cause of poisoning, out of which organophosphorus poisoning was 139 (17.39%), carbamate47 (5.88%) and organocholorine was 59 (7.38%) cases. Next common poisoning cause was drugs like diazepam, alprazolam, crocin, iron, were most commonly

used. Out of 799 cases drug consisted 121 cases (15.14%). Out of 799 patients 163 (20.40%) were expired in study period. Maximum number of people died because of aluminium phosphate (31.4%) followed by opc (20%) and rodenticide (9.6%). They concluded that the pesticides were the major cause of poisoning; the reasons are agriculture based economy, poverty and easy availability of highly toxic pesticides in India. The poisoning related mortality could be decreased by improving icu bed condition and appropriate supportive care at medical college and general hospital [44].

***AMARNATH M* (2012):** Analysed epidemiological study of medico legal organophosphorus poisoning in central region of nepal. Organophosphorus (op) pesticide self-poisoning is an important clinical problem in rural regions of the developing world. Organophospherous compounds are chemical compounds containing carbon-phosphorus bonds, primarily used in pest control and are often persistent organic pollutants. They are easily accessible, thus they are a commonly associated with suicides and accidental poisoning in nepal. Author's concluded the mean age group was 28 years, was prone to most of the cases. Most of the admitted cases were of suicidal as well as accidental in nature and women were the main victim than children. Suicidal deaths due to ingestion of op compound are very common in nepal, especially in women. The reason may be the increasing stress in the family and economic constraints. Accidental deaths due to occupational exposure or inhalation of op compounds are reported but in these cases mortality rate is less than that suicidal poisoning. Further study should be needed by government and national and international ngo to evaluate [45].

***SANJEEVKUMAR C* (2013):** Studied an epidemiological study of fatal aluminium phosphide poisoning at rajkot.Major occupation in saurashtra region of gujarat is farming with majority of population living in rural areas where the cases of accidental and suicidal poisoning are common and incidences are increasing day by day due to the use of pesticides for a wider variety of purposes. A detailed knowledge about the nature and magnitude of the poisoning cases in this particular area is not only important for early diagnosis and prompt treatment but also it may help to form policies to curb the access of the population to certain very toxic substances. The present study was undertaken in the department of forensic medicine at rajkot (gujarat) to know the pattern of fatal poisoning. Total 208 cases of death due to fatal poisoning were selected for this prospective study, which were brought to us for postmortem examination during the span of one year (from January 2007 to December 2007).

Various aspects like relation with age, sex, kind of poison, hospitalization, caste and time between consumption and death were noted in 208 cases of poisoning. Our study revealed that most of the victims of fatal poisoning were Hindus, married males of middle socio-economic status who died due to self-ingestion of some poison. Male: female ratio was found to be 1.36:1. In the present study higher number of fatal poisonings episodes can be ascribed to pesticides but on the basis of WHO classification of poison, aluminium phosphide is most common fatal poison [46].

AIM

The aim of this study is to collect the information about acute and chronic poisoning cases from various regions of south India(various hospitals located at Erode and Tirupure), also identify thevariation in treatment modality of acute and chronic poisoning cases by analysing method of treatment provided to the respective cases and observing clinical laboratory parameters, along with the identification of poisoning metabolites by using qualitative, quantitative and analytical toxicological assessment.

OBJECTIVE

- List out the common poisonous materials.
- Collect the common poison cases from the emergency & toxicology wards from various regions our selected hospital located at Erode and Tirupure.
- Collect the data about the mode of poisoning & nature of poisoning.
- Collect the data about the reference compounds & reagent needed for the qualitative analysis.
- Collect the data about instruments & cost needed for the quantitative analysis.
- Collect the data about the common treatments give to the patients.

PLAN OF WORK

The present study is focused to collect the information about qualitative & quantitative analytical methods of poisons & it's metabolites from various acute and chronic poison cases collected from Erode and Tirupure district of Tamil Nadu. The present study was planned to conduct in different toxicological emergency ward containing hospitals such as govt. District Headquarters Hospital Tirupur-642206, govt: District Headquarters Hospital, Erode-638112, Dhanvantri Critical Care Hospital poonkundranar street, Karunganlpalayam, Erode-638003.

➢ The Plan of Work Includes

- To collect the various poisoning cases.
- To collect relevant data for the investigations.
- To design a patient pro-forma.
    - Pro-forma containing patient details
    - Patient laboratory investigations
    - Vital signs and symptoms
    - Detail about poisons
    - Treatment modality

This information's included in the pro-forma form

- To design toxicology work sheet form.
    - Work sheet contains patient name, assay requested, sample type, laboratory investigations
    - Patient vital sing and symptoms, laboratory number
    - Qualitative analysis methods,
    - Test performed date, time, and signature of the analyst
    - Patients address

This information's included in the worksheet form.

To evaluate the collected data on the basis of age, sex, mode of poisoning, curability & mortality wise distribution area wise distribution, etc.

Data analysis was done with help of Microsoft.Excel-2010.

PATIENTS AND METHODOLOGY

- **STUDY SITE–**

  - Govt. District Head Quarters Hospital, Tirupur - 642206.
  - Govt. District Head Quarters Hospital, Erode- 638112.
  - Dhanvantri Critical care hospital poonkundranarnstreet, karungalpalayam, Erode-638003.

- **DURATION OF STUDY** - 6 Months.

- **STUDY POPULATION** - From the above said study sites 54 cases were collected, cases like pesticides, rodenticides, cow dung powder, oleander, alcohol intoxification organo-phosphorous, drugs, including snake bite. This study mainly based on the mode of poisoning, nature of poisoning & the analytical methods used for the diagnostic purpose also the information about the reagents, instruments, cost etc. needed for the analytical techniques.

- **<u>Following observations were made</u>**

- **Age**
- **Sex**
- **Address**
- **Mode of Poisoning**.[Acute poisoning due to suicidal & accidental exposure causes significant morbidity & mortality.]

- **Nature of Poisoning** [Pattern of poisoning in a region depends upon various factors such as availability, cost and access to toxic agents, socioeconomic status, cultural and religious characteristic of people. eg. Agricultural Poisoning, Rodenticides, cowdung powder, oleander seed, snake bite, pesticides, Varnish, alcohol intoxification, drugs etc.]

- **Analytical techniques** used for the Identification of poisons. [Qualitativeanalysis - it includes color tests, Quantitative analysis -it is used for identification as well as estimation of its concentration in the body.

- **Reference compounds** & **reagents** needed for the qualitative analysis. [For each tests different reference compounds and reagents needed].

  Reference compounds - Amo barbital, acetyl salicylic acid, caffeine, pentazocine, quinine, rhein. Etc.

  Reagents & solvents- Acetaldehyde, acetamide, acetic acid, barbituric acid, bromine, etc.

- **Instrument's & cost** needed for the quantitative analysis.[Toxicology laboratories use several methods to screen for poisons drugs, since there is no single,accurate, inexpensive method for this purpose. Each method differ in cost, accuracy, complexity, speed, and specificity. ***Instruments***- UV spectrophotometer, thin layer chromatography plates, HPLC, Mass-spectrometry and Gas-chromatography.

- ***Cost***- Drug abuse screen (Opioids, barbiturates, cocaine, cannabis)- Rs.1730

  Toxscreen- RS.1500, Pesticides– Rs.750.

- **Common treatments** given to the patients. [Activated charcoal- It binds to the poison and stops it from being further absorbed in the blood.

  Anti-dotes– This prevents the poison from working or reverse effects of the poison.

  Sedatives- If the person is agitated sedatives will give.

  A ventilator (Breathing machine) - This may be used if the person stops breathing.)

RESULT & ANALYSIS

GENDER WISE DISTRIBUTION

Table 5: Showing Gender Wise Distribution of Poisoning.

Gender	Number Of Patients	Percentage
Male	33	61%
Female	21	39%
Total	54	100%

In this study take 33 male and 21 female patient were taken for this study. In this study out of 54 patient 33(61%) patient were males and 21(39%) patient were females. According to this study male victims are more than the females.

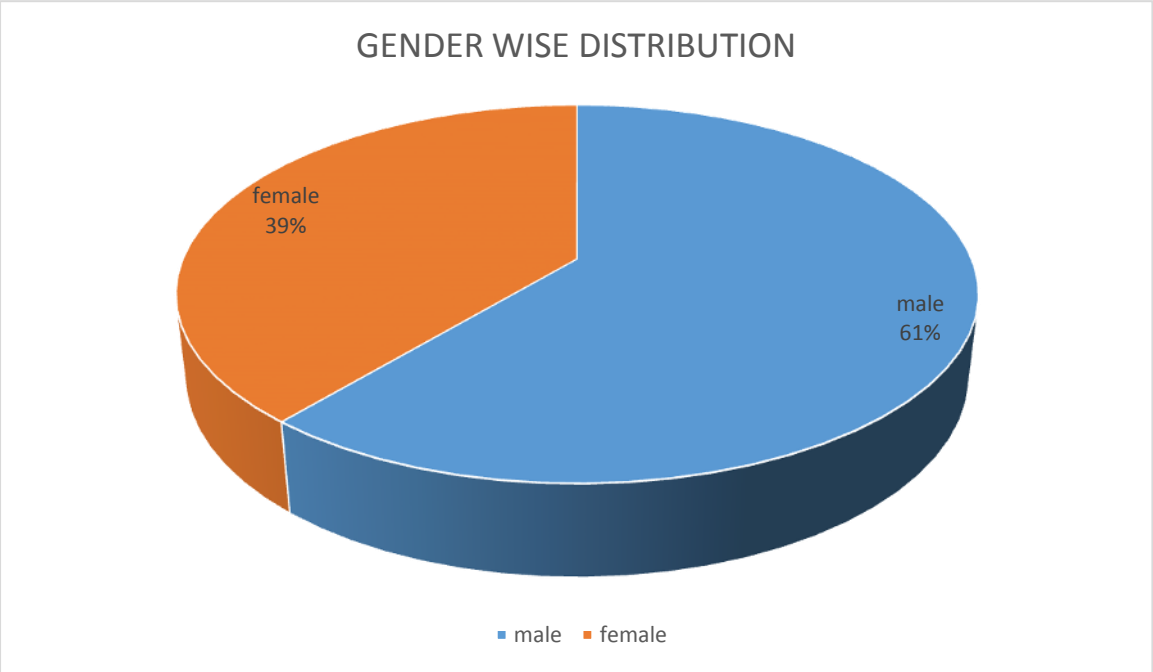


Figure 22: Illustrating Percentage of Gender Wise Distribution of Poisoning Cases.

AGE WISE DISTRIBUTION

Tablet 6: Showing Age Wise Distribution Of Poisoning Patient.

Age Of Patient	Male	Female	Total Number Of Patient	Percentage
15-25 years	6	6	12	22%
26-35 years	9	10	19	35%
36-45 years	7	0	7	13%
46-55 years	5	2	7	13%
56-65 years	6	3	9	17%

In this age wise study show the maximum number of patient belongs age between 26-35 (35%) years. The female patients were the major victims followed by the age group of 15-25 (22%).

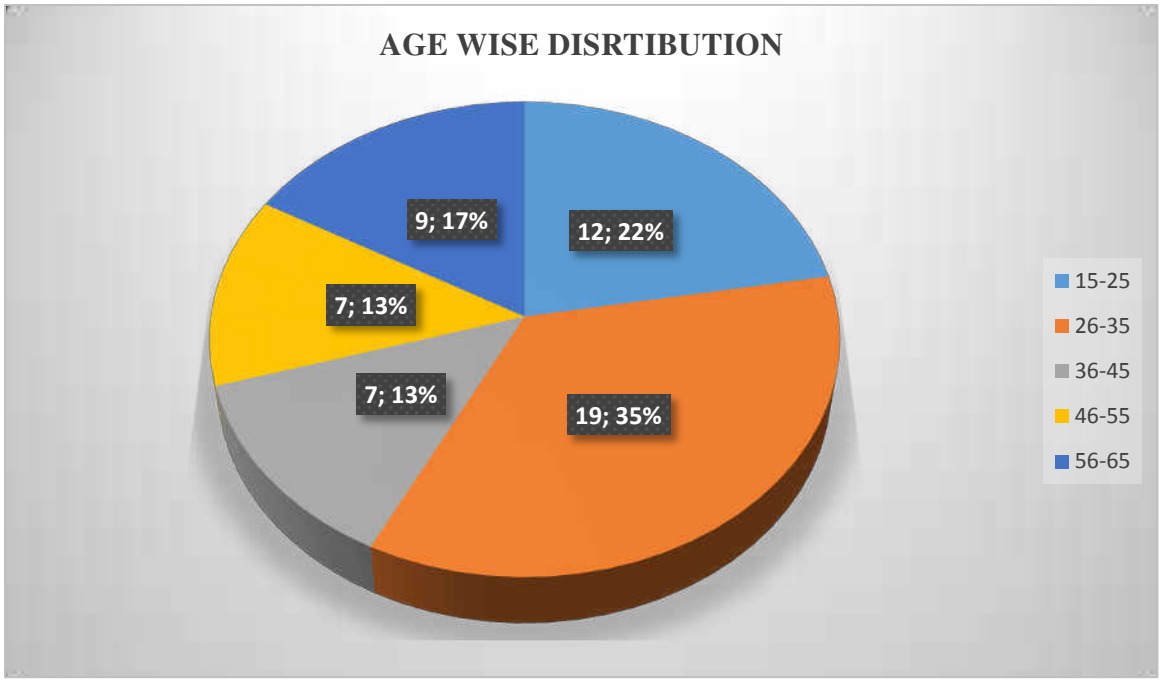


Figure 23: Illustrating the Percentage of Age Wise Distributions.

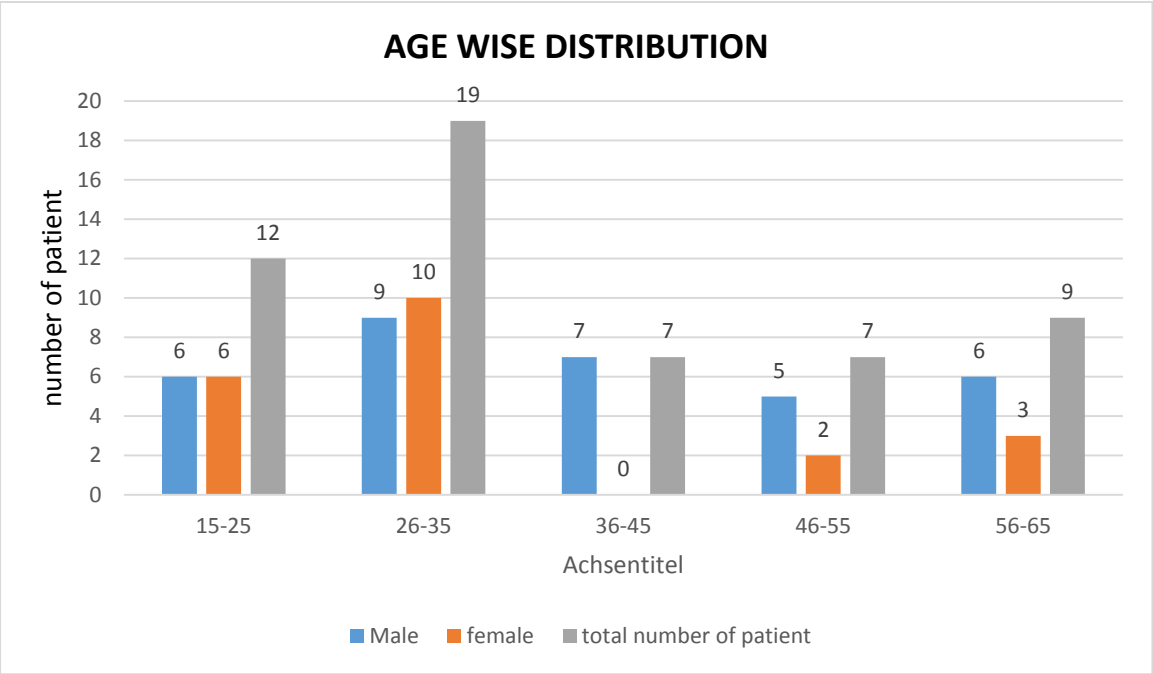


Figure 24: Illustrating the Percentage of Age Wise Distribution.

MODE OF POISON WISE DISTRIBUTION

Table 7: Showing Mode of Poisoning Cases.

Cases	Number of Patients	Percentage
Suicidal	37	69%
accidental	17	31%
Total	54	100%

Mode of poisoning wise study showing that the most majority of poisoning cases were belonging to suicidal (69%) followed by accidental poisoning cases (31%).

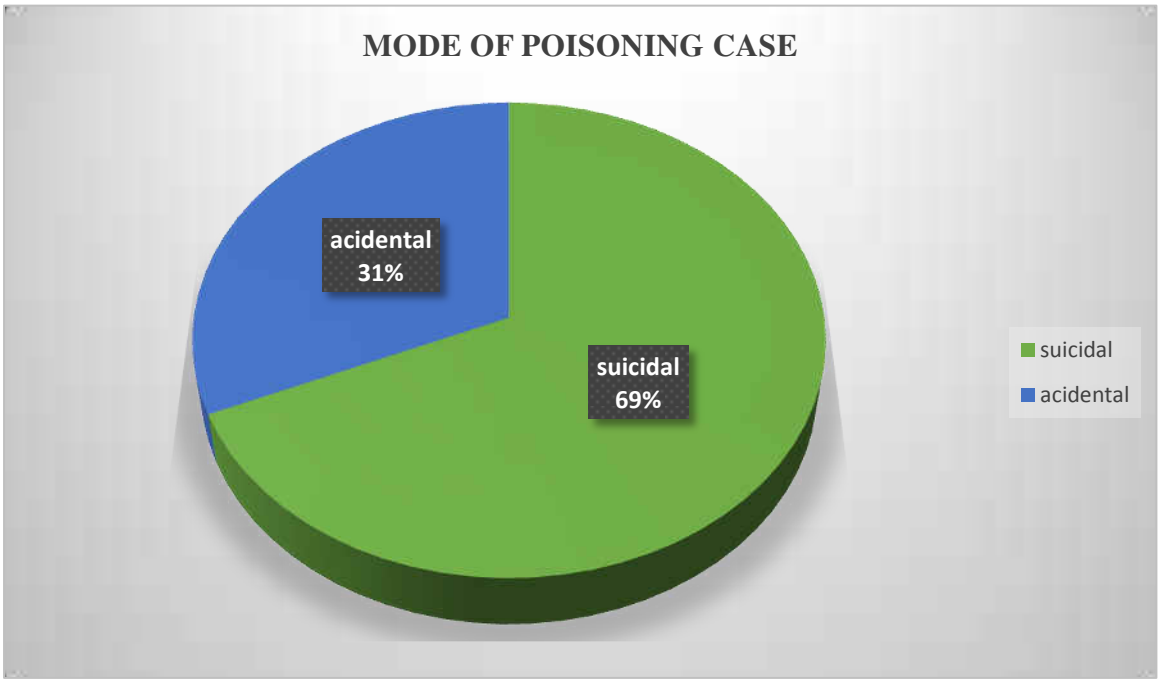


Figure 25: Illustrating Percentage Mode of Poisoning Cases.

NATURE OF POISON WISE DISTRIBUTION

Table 8: Showing Nature of Poison Wise Distribution in Poisoning.

Type of poisons	Male	Female	Total number of patient	Percentage
Monochrotophos	5	3	8	15%
Snake Bite	9	6	15	28%
Aluminium Phosphate	9	0	9	16%
Opcp	2	5	7	13%
Ant Killer	1	0	1	2%
Acid Poisoning	1	0	1	2%
Dermacron	1	0	1	2%
Varnish Poisoning	2	0	2	4%
Trichrophose	0	1	1	2%
Oleander Sead	0	1	1	2%
Scorpion	0	1	1	2%
Tablet Poisoning	0	1	1	2%
Lizole	0	1	1	2%
Ummet Poisoning	1	1	2	4%
Ratol Poisoning	2	1	3	5%
Total	33	21	54	100%

The overall poisoning cases study showing that the snake bit was the major cases of acute poisoning (28%) followed by aluminium phosphate (16%), ect.

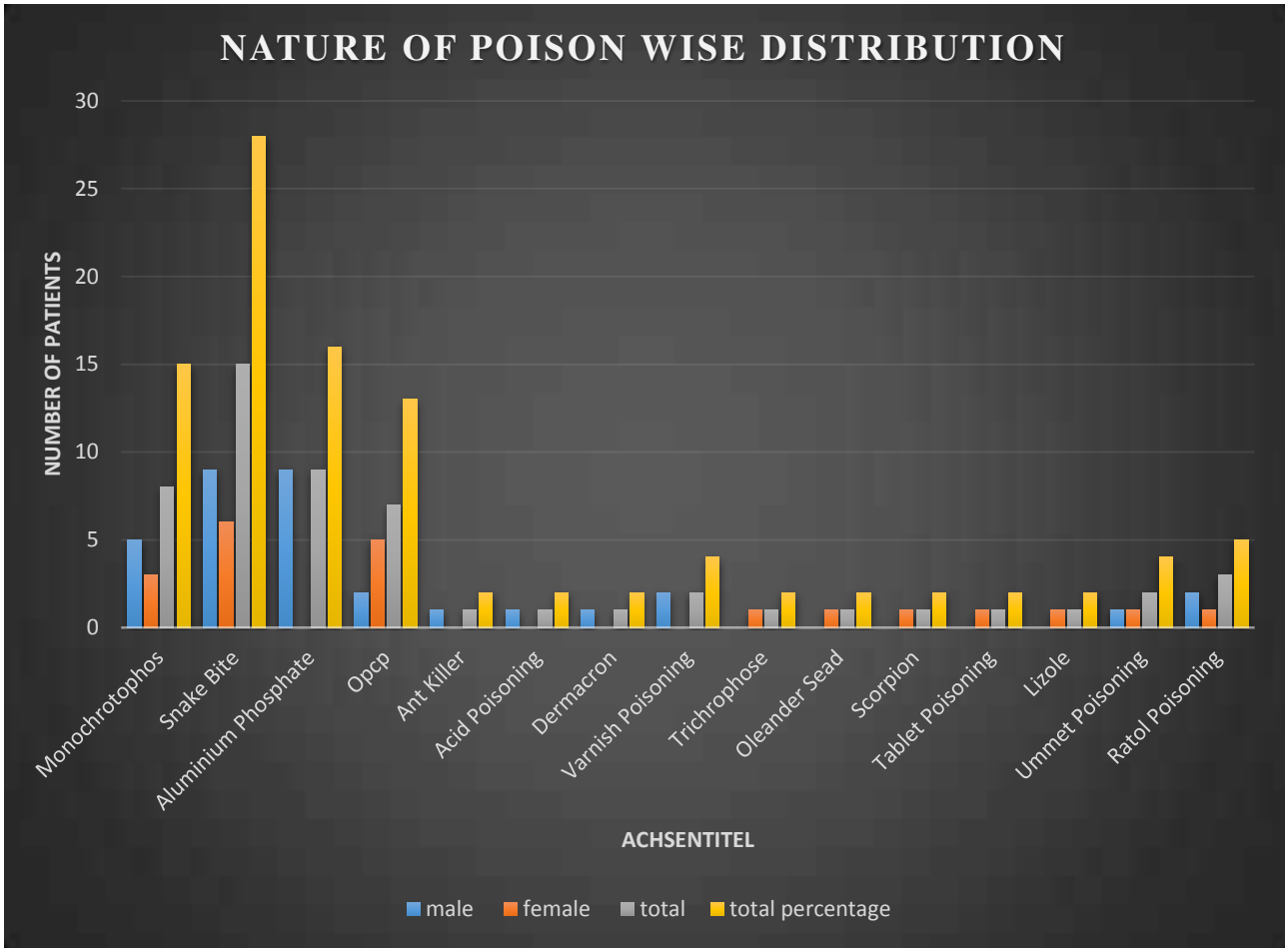


Figure 26: Illustrating Nature of Poison Wise Distribution in Poisoning Case.

SEX WISE CURABILITY

Table 9: Showing Sex Wise Curability Distribution.

Sex	Total Number Of Patient	Number Of Patient Cured	Percentage Of Curability	Number Of Patient Expired	Percentage Of Expired
Males	33	26	78.78%	7	21.21%
Females	21	18	85.71%	3	14.29%

This sex wise curability study shows most curable gender is female 85.71% (18) cases followed by male 78.78% (26).

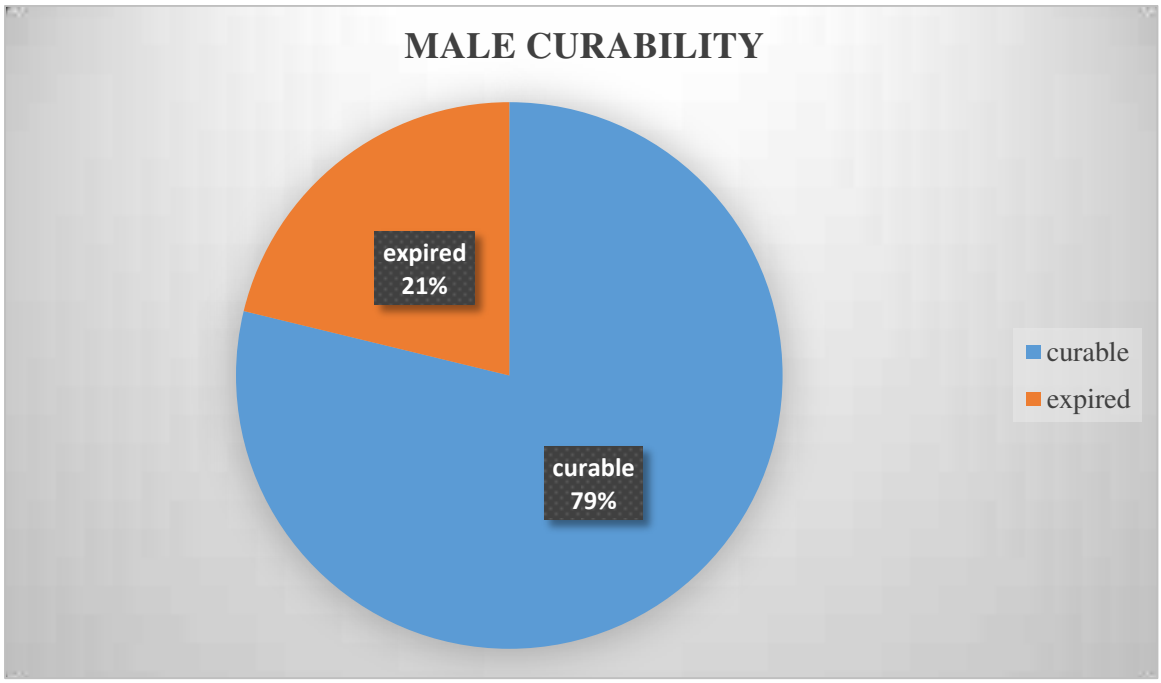


Figure 27: Illustrating Sex (males)Wise Curability.

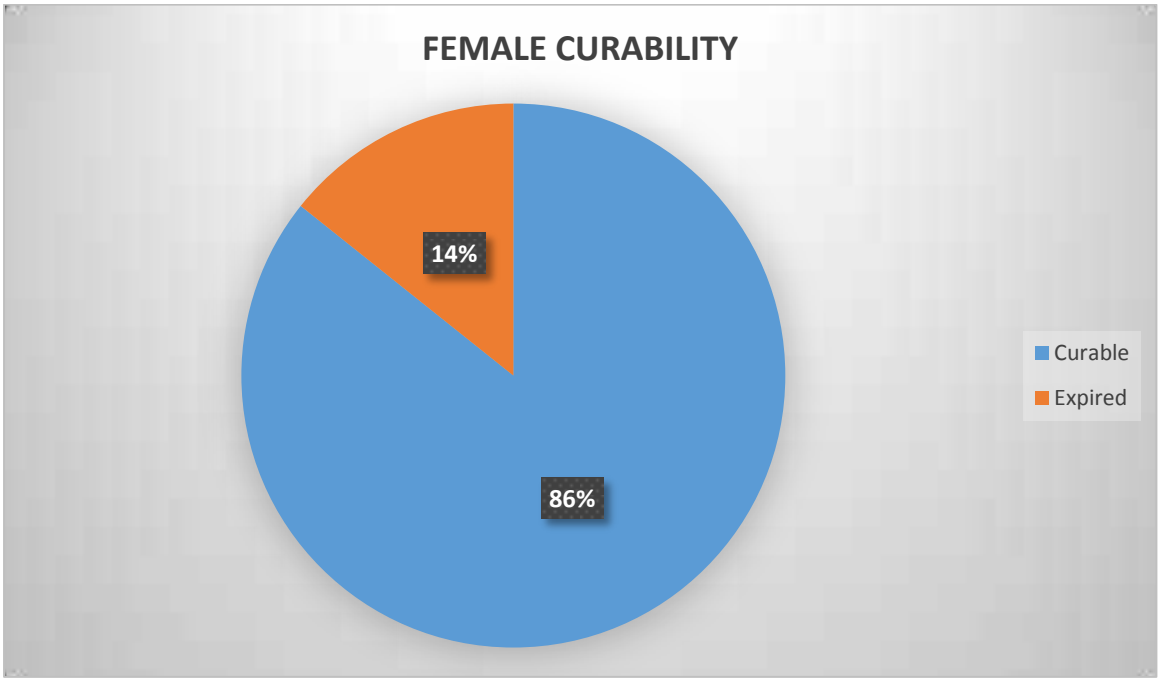


Figure 28: Illustrating Sex (females)Wise Curability.

DURATION OF TREATMENT

Chart 10: Showing Duration of Treatment for Male and Female.

Time Of Duration	Total Number Of Patient	Number of patient percentage	Male	Female
1-5 days	21	44%	11	10
6-10 days	24	39%	16	8
11-15days	7	13%	4	3
16-30days	2	4%	1	0

In this study shows that the most poisoning cases (44%) requires (1-5) days' time duration for recovery, followed by the case (39%) requires (6-10) days' time duration for recovery.

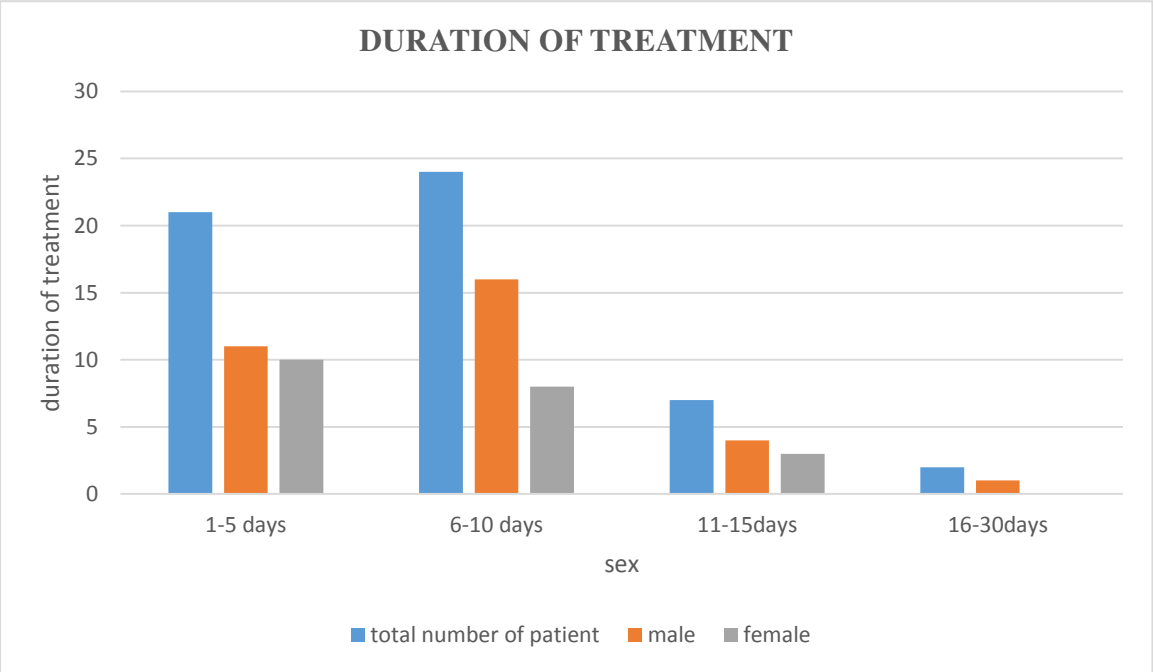


Figure 29: Illustrating Duration of Treatment for Male and Female.

CURABILITY AND MORTALITY WISE DISTRIBUTION

Table 11: Showing Curability and Mortality Wise Distribution of Poisoning Case.

Category	Number of patient	Percentage
Patient Cured	44	81%
Patient Expired	10	19%
Total	54	100%

curability and mortality wise distribution study showing that the curable patients was major victim (81%) and followed by motility (19%).

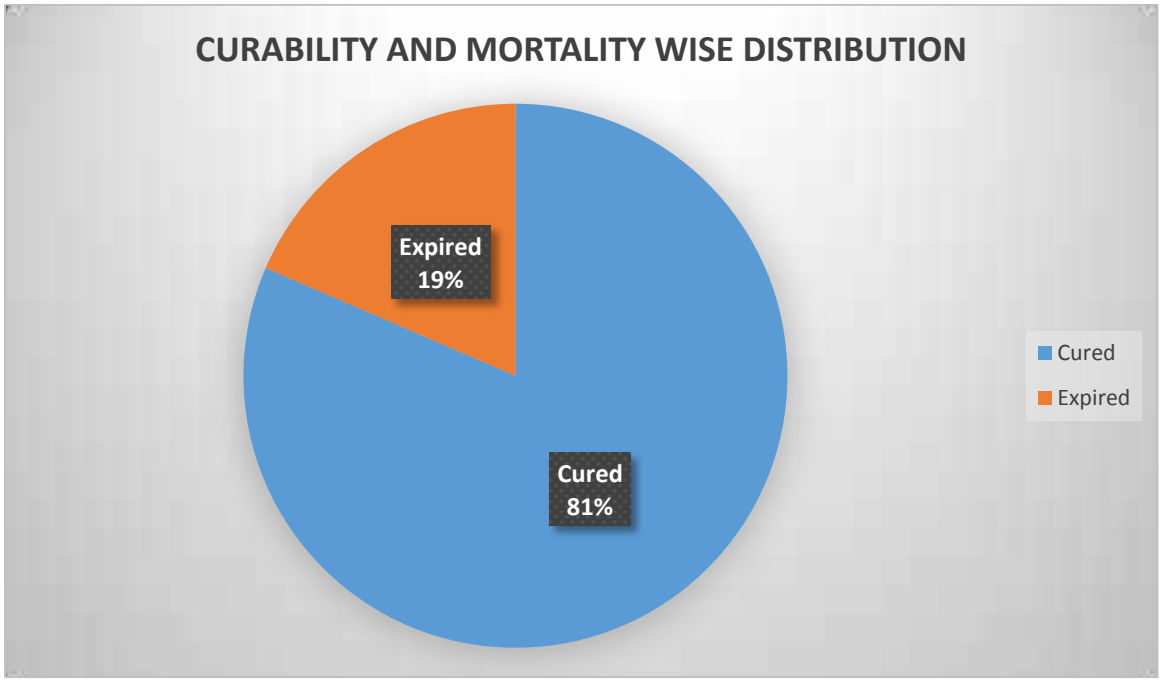


Figure 30: Illustrating Curability and Mortality Wise Distribution.

AREA WISE DISTRIBUTION

Table 12: Showingthe Area Wise Distribution of the Poisoning Cases.

Area	Number Of Patient	Percentage
Rural Area	38	70%
Urban	16	30%

The above study showing that the rural area poisoning cases were major 70%followed by urban area (30%).

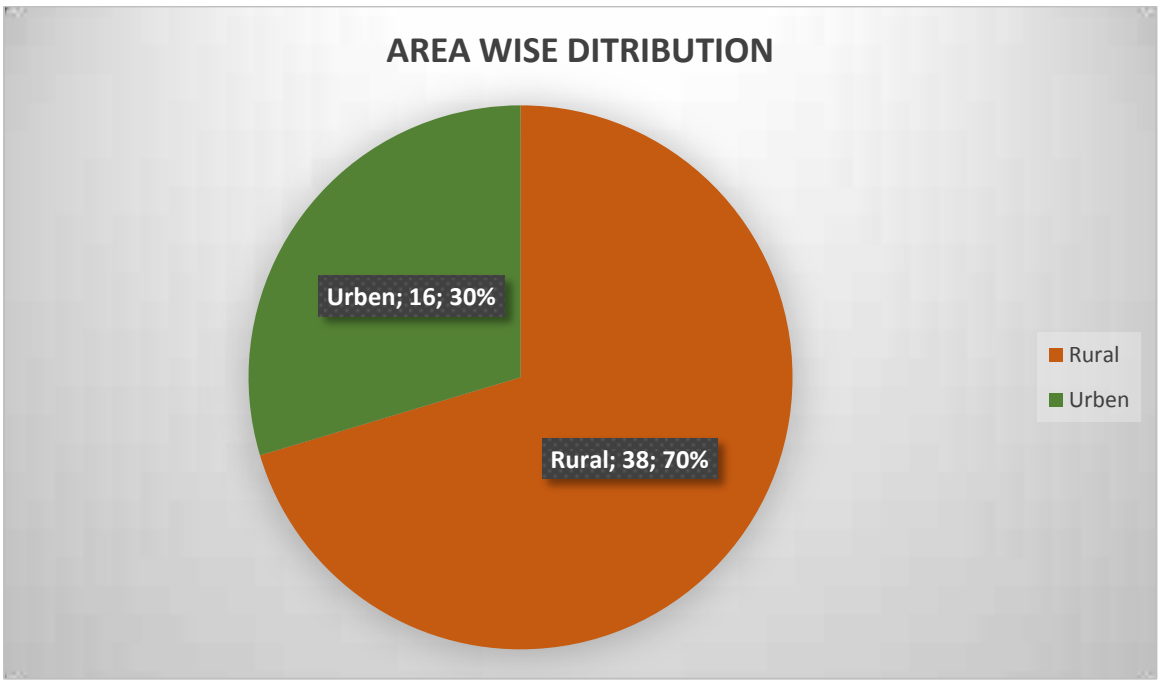


Figure 8: Illustratingthe Area Wise Distribution.

QUANTITATIVE ANALYSIS

Quantitative analysis refers to the determination of how much of a given component is present in a sample. The quantity may be expressed in terms of mass, concentration, or relative abundance of one or all components of a sample.

In quantitative analysis specialized equipment is needed and those methods are quite expensive.

- High Performance Liquid Chromatography (HPLC)
- Gas Chromatography (GC)
- Mass Spectrometry (MS)
- Radio Immuno Assay (RIA)
- Enzyme Mediated Immuno Assay Technique (EMIT)
- U.V Spectroscopy
- Infra red Spectroscopy
- Atomic Emission Spectroscopy (AES)
- Atomic Absorption Spectroscopy (AAS)
- Inductively Coupled Plasma Atomic Emission Spectroscopy (ICP-AES)
- Neutron Activation Analysis (NAA), etc.,

Table 13: Toxicological Analytical Tests, Methods, Reporting Time and Cost.

	Test Name	Specimen	Method	Assay	Reporting Time	Cost (Rs)
1	**Drug Abuse Screen** 1. Amphetamine 2. Methamphetamine 3. Opioids 4. Barbiturates 5. Phencyclidine, 6. Benzodiazepines 7.Cocaine 8. Cannabis/THC 9.Ketamine	Urine (15 ml)	ChromatographyImmuno assay	Qualitative	Same Day	1730
2	**Toxscreen**(Common representatives from each group) 1. Alcohols 2. Analgesics 3. Sedatives 4. Opioids 5.Pesticides 6.Phenothiazines	Blood (10 ml) Urine (50 ml Gastric Aspirate (10-25 ml)	Colour tests Chromatography Spectrophotometry	Qualitative	Same Day	1500
3	**Toxicology Screening Stage II** 1. Alcohols 2. Analgesics 3. Sedatives 4. Opioids 5. Phenothiazines	Blood (Plain 4-10 ml)Urine (50 ml)Gastric Aspirate (10-25 ml)	Colour tests Chromatography Spectrophotometry	Qualitative	Same Day	1150

4	**Toxicology Screening Stage I** 1. Alcohols 2. Analgesics 3. Opioids4. Phenothiazines	Blood (Plain 4-10 ml)Urine (50 ml)Gastric Aspirate (10-25 ml)	Colour tests Chromatography Spectrophotometry	Qualitative	Same Day	580
5	**Pesticides** 1. Aluminium Phosphide 2. Pyrethroids 3. Common Organophosphates 4. Diquat5. Organochlorines6. Paraquat 7. Carbamates 8. Zinc Phosphide	Blood (4-10 ml)Urine (50 ml)Gastric Aspirate (10-25 ml)	Colour tests Chromatography Spectrophotometry	Qualitative	Same Day	750
6	**Pesticides + Cholinesterase**	Blood (Plain 4-10 ml)Urine (50 ml)Gastric Aspirate (10-25 ml)	Chromatography Spectrophotometry	Qualitative / Quantitative	Same Day	1150
7	**Metals Screening** (With Quantification) 1. Antimony2. Arsenic 3. Bismuth4. Copper 5. Lead 6. Mercury	Blood (EDTA 4-10 ml) Commercial Products	ICP-AES	Quantitative	14 Days	1000
8	**Metals Urine Screening** 1. Antimony 2. Arsenic 3. Bismuth4. Copper 5. Lead 6. Mercury	Urine (50 ml)	Colour tests Spectrophotometry	Qualitative	Same Day	580

9	**Alcohols and Volatiles** 1. Ethanol 2. Methanol 3.Isopropanol 4.Ethylene glycol 5. Acetone 6. Chloroform 7. Carbon tetrachloride	Blood 10 ml Urine (50 ml) Gastric Aspirate (10-25 ml)	Colour tests Spectrophotometry	Qualitative	Same Day	750
10	**Inhalant Gases** 1. Carbon monoxide 2. Cyanides	Blood (4-10 ml)Urine (50 ml)Gastric Aspirate (10-25 ml)	Colour tests Spectrophotometry	Qualitative	Same Day	750
11	**Sedatives** 1. Common Barbiturates 2. Common Benzodiazepines3. Chloral hydrate	Blood 4-10 ml)Urine (50 ml)Gastric Aspirate (10-25 ml)	Colour tests Chromatography Spectrophotometry	Qualitative	Same Day	750
12	**Narcotics** (Opioids) 1. Buprenorphine 2. Codeine 3.Dextromethorphan4. Fentanyl 5. Morphine 6. Noscapine 7. Pentazocine8. Tramadol 9. Dextro propoxyphene	Blood (4-10 ml)Urine (50 ml)Gastric Aspirate (10-25 ml)	Colour tests Chromatography Spectrophotometry	Qualitative	Same Day	750
13	**Anticholinergics**1. Atropine2. Antihistamines (Common) 3. Scopolamine	Blood 4-10 ml)Urine (50 ml),Gastric Aspirate (10-25 ml)	Colour tests Chromatography Spectrophotometry	Qualitative	Same Day	750

14	**Anticholinergics**1. Atropine 2. Antihistamines (Common) 3. Scopolamine 4.Phenothiazines5. Cyclic antidepressants (Common)	Blood 4-10 ml)Urine (50 ml)Gastric Aspirate (10-25 ml)	Colour tests Chromatography Spectrophotometry	Qualitative	Same Day	750
15	**Antimicrobials** (Common drugs)1. Aminoglycosides 2. Metronidazole 3. Tinidazole 4. Penicillins5. Cephalosporines 6. Macrolides 7. Tetracyclines	Blood (4-10 ml)Urine (50 ml)Gastric Aspirate (10-25 ml)	Chromatography Spectrophotometry	Qualitative	Same Day	750
16	**Analgesics and Anti-inflammatories** 1. Salicylates2. Paracetamol3. Selected NSAIDs	Blood (4-10 ml)Urine (50 ml)Gastric Aspirate (10-25 ml)	Colour tests Chromatography Spectrophotometry	Qualitative	Same Day	750
17	**Rodenticides** (Rat poisons) 1. Zinc phosphide 2. Phosphorus 3. Long acting anticoagulants	Blood (4-10 ml)Urine (50 ml)Gastric Aspirate (10-25 ml)	Colour tests Chromatography Spectrophotometry	Qualitative	Same Day	750
18	**Steroids** 1) Cortisone 2) Hydrocortisone 3) Prednisone 4) Prednisolone 5) Betamethasone 6) Dexamethasone	Blood (4-10 ml)Urine (50 ml)Gastric Aspirate (10-25 ml)	Colour tests Chromatography Spectrophotometry	Qualitative	Same Day	870

19	**Cholinesterase level**	Blood	Spectrophotometry	Quantitative	Same Day	610
20	**Antipsychotics**1) Risperidone 2) Imipramine 3) Carbamazepine 4) Fluoxetine, 5) Escitalopram6) Lithium7) Haloperidol 8) Olanzapine9) Quetiapine10) Fluroxamine11) Paroxetine 12) Sertraline	Blood (4-10 ml)Urine (50 ml)Gastric Aspirate (10-25 ml)	Colour tests Chromatography Spectrophotometry	Qualitative	Same Day	950
21	**Common Barbiturates**	Blood (4-10 ml) Urine (50 ml)Gastric Aspirate (10-25 ml)	Chromatography Immunoassay	Qualitative	Same Day	500
22	**Common Benzodiazepines**	Blood (4 ml)Urine (50 ml), Gastric Aspirate(10-25 ml)	Chromatography Immunoassay	Qualitative	Same Day	500
23	**TCA (Tricyclic antidepressants)**1) Amitriptyline2) Clomipramine 3)Promazine4) Cyclobenzaprine 5) Doxepin 6) Imipramine 7) Trimipramine8) Desipramine 9) Nortriptyline10) Protriptyline 11)Nordoxepin12) Perphenazine	Blood (Plain 4-10 ml)Urine (50 ml)Gastric Aspirate (10-25 ml)	Chromatography	Qualitative	Same Day	500

Table 14: Type of Poisons with its vernacular name (Tamil & Malayalam) and its Chemical Constituent.

	Type Of Poison	Vernacular Name (Tamil)	Vernacular Name (Malayalam)	Chemical Constituent	Specimen Used To Detect	Determination Technics	Cost Of Test
1	**Pesticides**	Poochikolli	Keedanashini	Aluminium Phosphide, Pyrethroids, Common Organophosphates, Diquat, Organochlorines, Paraquat, Carbamates, Zinc Phosphide	Blood (4-10ml) Urine (50ml) Gastric Aspirate (10-25 Ml)	Colour Tests Chromatography Spectrophotometry (Qulitqtive)	750
2	**Opcp** [Organophosphorous Compound]			Aluminium Phosphide, Zinc Phosphide, etc..	Blood (4-10ml) Urine (50ml) Gastric Aspirate (10-25 Ml)	Colour Tests Chromatography Spectrophotometry (Qulitqtive)	750
3	**Monocrotophos**			Monocrotophous	Blood (4-10ml) Urine (50ml) Gastric Aspirate (10-25 Ml)	Colour Tests Chromatography Spectrophotometry (Qulitqtive)	750
3	**Snake Venom**	PambuVisham	SarpaVisham	Neurotoxin(Metalloproteinase), Polypeptide Toxin(,Cardio Toxins, Cytotoxins,) Eg:Calalase, Dehydrogenase Lactate, Hyaluronidase, Fibrinogenase, Heparinise, Elastase	Blood (4-10ml) Urine (50ml)	Blood Coagulation Plate Late Count	450

4	**Aluminium Phosphate**			Aluminium Phosphate	Blood (4-10ml) Urine (50ml) Gastric Aspirate (10-25 Ml)	Colour Tests Chromatography Spectrophotometry (Qulitqtive)	750
5	**Ant Killer**	ErumbbuMarundhu	ChayiPodi	Organochlorine (Ddt, Aldrin, Dieldrin,MalaThion)	Blood (Plain 4-10 Ml) Urine (50 Ml) Gastric Aspirate (10-25 Ml)	Colour Tests Chromatography Spectrophotometry (Qualitative	750
6	**Acid Poisoning** [Sulphuric Acid]	Sulphuric Acid	Sulphuric Acid	Sulphuric Acid	Vomiting content	Chromatography, litmus, Spectrophotometry (Qualitative)	500
7	**Di chromium Bromide Poisoning**	DCB	DCB	Di Chromium Bromide	Blood (4-10ml) Urine (50ml) Gastric Aspirate (10-25 Ml)	Colour Tests Chromatography Spectrophotometry (Qulitqtive)	750
8	**Varnish Poisoning**	Varnish	Varnish	Resin(Amber,Baisam,Rosin) Solvent(Ethanol,MineralSpirit, Turpentine)	Blood (4-10ml) Urine (50ml) Gastric Aspirate (10-25 Ml)	Colour Tests Chromatography Spectrophotometry (Qulitqtive)	750
9	Tricrophose			Tri Cromiun Phosphate	Blood (4-10ml) Urine (50ml) Gastric Aspirate (10-25 Ml)	Colour Tests Chromatography Spectrophotometry (Qulitqtive)	750

10	**Rodenticides** (Rat Poisons)	Yelivisham	Yelivisham	Zinc Phosphide, Phosphorus Long Acting Anticoagulants	Blood (4-10ml) Urine (50ml) Gastric Aspirate (10-25 Ml)	Colour Tests Chromatography Spectrophotometry (Qulitqtive)	750
11	**Lizole**	Lizol	Lizol	Phenol,IsopropylAlcohol,PotassiumHydroxide,,PhenylPhenol, Ethanol	Blood (4-10ml) Urine (50ml) Gastric Aspirate (10-25 Ml)	Colour Tests Chromatography Spectrophotometry (Qulitqtive	750
12	**Datura**	Umattai, Umatta, Amittai, Uma Thai	Ummath	Tropane Alkaloids(Scopolamine, Hyoscyamine, atropine.)	Blood (4-10ml) Urine (50ml) Gastric Aspirate (10-25 Ml)	Colour Tests Chromatography Spectrophotometry (Qualitative	750
13	**Tablet Poisoning** [Parcetamol]				Blood (4-10ml) Urine (50ml) Gastric Aspirate (10-25 Ml)	Colour Tests Chromatography Spectrophotometry (Qulitqtive	750
14	**Oleander Seed**	AraliVidhai	Aralikazha	Oleandrin, Oleandrigenin,Thevetin A, Thevetin B.	Blood (4-10ml) Urine (50ml) Gastric Aspirate (10-25 Ml)	Colour Tests Chromatography Spectrophotometry (Qulitqtive	750

METHODS OF TREATMENT TOWARDS DIFFERENT POISONS

Based on our observation, it was reviewed that there different kind of poisons cases were treated according to the following methods with respect to condition of the patients and severity of illness as follows.

1. ALUMINIUM PHOSPHATE POISONING SIGNS, SYMPTOMS, AND TREATMENT.

Sign And Symptoms	Therapy
➢ Hypotension (refractory to dopamine therapy). ➢ Dizziness, Fatigue, Tightness in chest. ➢ Head ache, Nausea , Vomiting ➢ Diarrhea, Ataxia, Numbness ➢ Paresthesia, Tremor, Muscle weakness. ➢ Diplopia, Jaundice. ➢ Acute respiratory distress syndrome. ➢ Heart failure, arrthymiasis, convulsion and coma. ➢ Late manifestation includes liver and kidney damage. ➢ Electrolytic imbalance. ➢ Fatty liver. ➢ Respiratory failure. ➢ Plasma glucose, serum urea, creatinine and liver enzymes showed a significant rise from normal level. ➢ Death may be due to combination of one or above complications.	➢ Symptomatic and supportive aid. ➢ Gastric lavage with KMnO4 (potassium permagnate). ➢ Stomachs wash withslurry of activated charcoal. ➢ Oxygen supply. ➢ MgSO4(magnesium sulfate) ➢ Midazolam(benzodiazepine) ➢ ceftriaxone ➢ clindamycin ➢ pantoprazole ➢ dobutamine ➢ Amikacin(aminoglycoside anti-biotic) ➢ furosemide ➢ Torsenamide ➢ Cefixime, moxifloxacin ➢ Folic acid ➢ ofloxacin ➢ vitamin B-complex

2. ORGANOPHOSPHOROUS POISONING SIGNS, SYMPTOMS, AND TREATMENT.

Sign And Symptoms	Therapy
➢ excessive accumulation of acetylcholine at the synapses ➢ miosis, bradycardia. ➢ Increased gastrointestinal motility. ➢ Emesis, sweating, tachypnea. ➢ Salivation, lacrimation, altered sensorium, fasciculation. ➢ Bronchospasm, blurred vision, urination and defecation. ➢ The complications include acidosis, respiratory paralysis ➢ Acute renal failure. ➢ Seizures, arrhythmia, aspiration. ➢ Electrolytic imbalance. ➢ Death may be due to combination of one or above complications. ➢ Plasma glucose, serum urea, creatinine and liver enzymes showed a significant rise from normal level.	➢ Stomach washes with Slurry of activated charcoal. ➢ Oxygen supply ➢ Cholinesterasereactivator, pralidoxime. ➢ Ceftriaxone ➢ Amikacin(amino glycocide) ➢ Pantoprazole ➢ ATSO4(atropine sulphate) ➢ Methyl prednisolone ➢ Folic acid ➢ Acetyl cysteen ➢ Viit B-complex ➢ ofloxacine

3. SNAIK BITE POISONING SIGNS, SYMPTOMS, AND TREATMENT.

Sign And Symptoms	Therapy
Chief complaints. ➢ Pain, Redness $ Swelling In The Area Of Bite ➢ Nausea (Feeling Sick) &Vomiting. ➢ Dizziness & Fainting. ➢ Blistering & Eventually, Gangrene In the Area ofthe Bite. ➢ Shock. ➢ Muscle Paralysis (An Inability To Move One Or More Muscles Of The Body) Leading To Difficulty, Swallowing &Breathing. ➢ Bleeding. ➢ Swelling of the lips, gums &tongue. ➢ Irregular heartbeat. **General symptoms.** ➢ Central. • Dizziness. • Fainting. • Increased thirst. • Headache. ➢ Vision. • Blurriness. ➢ Systemic.	➢ O_2supply ➢ ASV(anti snake venom) ➢ HCO3(bi-carbonate) ➢ TT(tetanus toxoid) ➢ Atropine. ➢ Gentamycin ➢ Neostigmine ➢ Ceftriaxone ➢ Ranitidine ➢ Gentamicin ➢ Cefepime ➢ Foli acid ➢ Vit B-complex ➢ Torsenamide ➢ Ofloxacin ➢ Pantoprazole ➢ Blood unit

• Fever. • Severe pain. ➢ Heart and vessels. • Rapid pulse. • Low blood pressure. • Severe shock. ➢ Respiratory. • Breathing ➢ Muscular. • Convulsions. • Loss of co- coordination. ➢ Wound site. • Bleeding. • Fang marks. • Discoloration. • Burning sensation. • Swelling. ➢ Gastric. • Nausea.	

4. RATOL POISONING (ZINC PHOSPHIDE) POISONING SIGNS, SYMPTOMS, AND TREATMENT.

Sign And Symptoms	Therapy
➢ Irritability, drowsiness. ➢ Tremors,vertigo,diplopia. ➢ Ataxia,cough,dyspnea. ➢ Retrosternal discomfort. ➢ Abdominal pain, and vomiting. ➢ Hypotension. ➢ Reducing cardiac output. ➢ Tachycardia, oliguria ➢ Anuria, cyanosis ➢ Pulmonary edema, tachypnea. ➢ Jaundice ➢ Hepatosplenomeglay ➢ ileus, seizures,	➢ Stomach washes with Slurry of activated charcoal orally. ➢ O_2supply ➢ HCO3(bi-carbonate) ➢ Noradrenalin ➢ Acetyl cysteine ➢ Methyl prednisolone ➢ Ampicillin ➢ Clindamycin ➢ Pantoprazole ➢ Vasopressin. ➢ Vitamin k ➢ KClpotassium chloride) ➢ Filgrastim

5. UMMMET POISONING SIGNS, SYMPTOMS, AND TREATMENT.

Sign And Symptoms	Therapy
➢ jaw breathing ➢ hypoxia ➢ sinus tachycardia ➢ photophobia ➢ mydriasis ➢ amnesia ➢ hyperthermia ➢ vomiting ➢ stomach Paine, head ache ➢ seizures	➢ Stomachs washwith slurry of activated charcoal. ➢ Oxygen supply ➢ Physostigmine ➢ Pancuronium bromide ➢ Midazolam ➢ Buprenorphine ➢ Cephalosporin ➢ Gentamicin ➢ pantoprazole ➢ pralidoxime ➢ Atropine ➢ Glycopyruvate ➢ Methyl prednisolone ➢ Budamate(formoterolfumerate, budesonide)

6. MONOCROTOPHOSE POISONING SIGNS, SYMPTOMS, AND TREATMENT.

Sign And Symptoms	Therapy
➢ Respiratory failure ➢ Fever ➢ Ventricular diastolic dysfunction ➢ Change in BP, PS, RS. ➢ Plasma glucose, serum urea, creatinine and liver enzymes showed a significant rise from normal level. ➢ GRF rate decreased ➢ Variation on electrolate balance ➢ Decreased Hb.	➢ Stomach wash Slurry of activated charcoal. ➢ O_2supply ➢ ALTSO4(atropine sulphate) ➢ Magnova (cefepime/tazobactam) ➢ ceftriaxone ➢ Gentamycin ➢ Pantoprazole ➢ Morphine ➢ Amlodipine ➢ Midazolam ➢ Iron supplement ➢ B-complex ➢ Palidoxime ➢ Glycol pyruvate ➢ Pancuronium bromide ➢ Methyl prednisolone ➢ Folic acid ➢ Multi vitamin

7. LIZOLE POISONING SIGNS, SYMPTOMS, AND TREATMENT.

Sign And Symptoms	Therapy
➢ Respiratory failure ➢ Fever ➢ Ventricular diastolic dysfunction ➢ Change in BP, PS, RS. ➢ Change pH. ➢ Loss of vision. ➢ Head ache. ➢ Seizures. ➢ Severe pain in throat. ➢ Sever pain or burning in nose,eyes,ears, lips or tongue. ➢ Abdominal pain, vomiting, vomiting blood, blood in stool ➢ Plasma glucose, serum urea, creatinine and liver enzymes showed a significant rise from normal level. ➢ Hypotension. ➢ GRF rate decreased ➢ Variation on electrolate balance ➢ Decreased Hb. ➢ Coma (decreased level of consciousness and lackof consciousness).	➢ Stomach wash Slurry of activated charcoal orally. ➢ O2 supply. ➢ ceftriaxone ➢ Amikacin (amino glycoside antibiotic) ➢ pantoprazole ➢ Ofloxacin ➢ B-Complex ➢ Methyl prednisolone

8. OLEANDER SEED POISONING SIGNS, SYMPTOMS, AND TREATMENT.

Sign And Symptoms	Therapy
➢ Respiratory failure ➢ Fever ➢ Nausea ➢ Vomiting ➢ Excess salivation ➢ Abdominal pain ➢ Diarrhea they may containing blood ➢ Drowsiness ➢ Tremor ➢ Shaking of muscles ➢ Seizer, collapse ➢ coma ➢ Ventricular diastolic dysfunction ➢ Change in BP, PS, RS. ➢ Plasma glucose, serum urea, creatinine and liver enzymes showed a significant rise from normal level. ➢ GRF rate decreased ➢ Variation on electrolyte balance ➢ Decreased Ph	➢ Stomach wash slurry of activated. ➢ O2 supply. ➢ Digoxin. ➢ Ceftriaxone ➢ Amikacin(amino glycocide) ➢ Glycopyruvate ➢ Paracetamol ➢ Folic acid ➢ Vit B-Clomplex ➢ Pantoprazole ➢ Ofloxacin ➢ Orciprenalin

9. SCORPION BITEPOISONING SIGNS, SYMPTOMS, AND TREATMENT.

Sign And Symptoms	Therapy
➢ Pain, redness and warmth at the stinging site. ➢ Tingling, Swelling around the sting site. ➢ Muscle twitching, sweating ➢ Vomiting ➢ Change in blood pressure. ➢ Accelerate heart rate ➢ Restlessness ➢ Blurry vision	➢ O_2supply ➢ Dexamethazone ➢ Prazosin ➢ Methyl prednisolone ➢ Ceftriaxone ➢ Amikacin(aminoglycoside) ➢ Pantoprazole ➢ Tramadol ➢ Folic acid ➢ Torasemide ➢ Ofloxacin ➢ Multivitamin

10.TRICROMIUN PHOPHATEPOISONING SIGNS, SYMPTOMS, AND TREATMENT.

Sign And Symptoms	Therapy
➢ Excessive accumulation of acetylcholine at the synapses ➢ Miosis, bradycardia. ➢ Increased gastrointestinal motility. ➢ Emesis, sweating, tachypnea. ➢ Salivation, lacrimation, altered sensorium, fasciculation. ➢ Bronchospasm, blurred vision, urination and defecation. ➢ The complications include acidosis, respiratory paralysisand acute renal failure. ➢ Seizures, arrhythmia, aspiration. ➢ Electrolytic imbalance. ➢ Death may be due to combination of one or above complications. ➢ Plasma glucose, serum urea, creatinine and liver enzymes showed a significant rise from normal level.	➢ Stomach wash Slurry of activated. ➢ O_2supply ➢ ceftriaxone ➢ Amikacin(aminoglycoside) ➢ Pantoprazole ➢ Pralidoxime ➢ Folic acid ➢ Vit B-complex ➢ ATSO4(atropine sulphate) ➢ Glycopyruvate ➢ Ofloxacin ➢ Multi vitamin

11.H_2 SO_4POISONING SIGNS, SYMPTOMS AND TREATMENT.

Sign And Symptoms	Therapy
➢ Gastric irritation ➢ Stomach pain ➢ Vomiting ➢ Head ache ➢ Burning of heart ➢ Ulcer in mouth and stomach ➢ Blood P_H change	➢ Ingestion of zinc chloride, mercuric chloride, or hydrogen fluoride. ➢ Stomach washes with Slurry of activated Charcoal. ➢ O_2supply ➢ ceftriazone ➢ Clintamycin ➢ Sucralfate ➢ Pantoprazole ➢ Methylprednisolone ➢ Folic acid ➢ Vit B-Comlpex ➢ Blood units

12.DEMACRONPOISONING SIGNS, SYMPTOMS, AND TREATMENT.

Sign And Symptoms	Therapy
➢ Left ventricular diastolic dysfunction ➢ High B.P ➢ High pulse rate ➢ Low respiratory rate ➢ Left ventricular dysfunction	➢ Stomach washes with Slurry of activated charcoal orally. ➢ O_2supply ➢ Morphine ➢ Pancuronium bromide ➢ Midazolam ➢ Furosemide ➢ Promethazine ➢ KCl(potassium cloride) ➢ Pralidixime ➢ ceftriaxone ➢ Amikacin(amino glycoside) ➢ Pantoprazole ➢ Folic acid ➢ Vit B-Complex ➢ Torsenamide

13.PARACETAMOLPOISONING SIGNS, SYMPTOMS, AND TREATMENT.

Sign And Symptoms	Therapy
➢ Gastric irritation ➢ Stomach pain ➢ Vomiting ➢ Head ache ➢ Burning of heart ➢ Low blood sugar ➢ Sweating ➢ Nausea ➢ vomiting ➢ Blood P_H change ➢ Anemia	➢ Stomach washes with Slurry of activated charcoal orally. ➢ O_2supply ➢ Acetyl cysteine ➢ Ofloxacin ➢ Pantoprazole ➢ Epidril ➢ Escitalopram ➢ Vit B-Complex ➢ Folic acid ➢ Multi vitamine

14.VARNISHPOISONING SIGNS, SYMPTOMS, AND TREATMENT.

Sign And Symptoms	Therapy
➢ Gastric irritation ➢ Stomach pain ➢ Vomiting ➢ Head ache ➢ Burning of heart ➢ Blood P_H change ➢ Sinus tachycardia ➢ Left ventricular diastolic dysfunction ➢ Arrhythmias ➢ Seizer ➢ Electrolyte imbalance	➢ Stomach washes with Slurry of activated charcoal orally. ➢ O_2supply ➢ Buprenorphine ➢ Ceftriaxone ➢ Gentamicin ➢ pantoprazole ➢ Mannitol ➢ Dexamethasone ➢ Saline ➢ Vit B-Complex ➢ Multi vitamin ➢ Folic acid ➢ Blood units

Summary

From this retrospective study was observed that, there is alarming increases of poisoning cases are mainly for suicidal purpose (69%). Agricultural poisons were found to be the most common causes of acute poisoning cases (55%). The major number of poisonings cases are reported in the young people's age range (26-35 years old) (35%) and followed by the peoples age range (15-25years old) (22%).

- Males were the major victims in overall poisoning cases (61%) and the most poisoning cases were from rural area than the urban area. Acute poisoning is also common reason for hospitalization and most poisoned patients make a full recovery without specific treatment .however, with some common poisons analytical toxicological data can be important in establishing a diagnosis and guiding the treatment. Most of the acute poisoning cases are treated against symptoms.
- Acute poisoning cases also included animal envenomation (29%). Animal envenomation cases males are major victims (58.82%), mainly comes from rural areas its due to males are spent more time in agricultural areas. Animal envenomation can be treated with anti-venom with supportive therapy. Some rare cases of snake envenomations may lead to chronic toxicity of organs.
- Chronic toxicity is the development of adverse effect as the result of long term exposure to a toxicant or other stressor. It can manifest as direct lethality but more commonly refers to sub-lethal endpoints such as decreased growth, reduce reproduction, or behavioral changes such as impacted swimming performance etc.
- Chronic poisoning are more difficult to evaluate and quantify, there insidious, symptoms, non-specific and sometimes multi organic manifestations, along with frequent lack of association with an external toxic agent hamper diagnosis, resulting in official figures, mostly involving, the work place, that undoubtedly underestimate the scope of problem.
- The availability of reliable analytical facilities can also assist in other clinical areas such as assessing illicit drug use and the diagnosis and treatment of poisoning with environmental toxins such as lead, as well as in the management of incidents related to the accidental or deliberate release of chemicals into the environment (chemical incidents) and other aspects of chemical safety.

- India is a predominantly agricultural region and pesticides and rodenticide are used control rodent and pest in agricultural fields. So the agricultural poisons freely available in market. That why the majority poisoning cases are coming from agricultural poisons. Most poisoning cases are reported from rural areas (70%).
- Aluminum phosphide, zinc phosphide, ethylene di bromide are widely used agricultural pesticides and rodenticides, such as mice and rats and also including voles, ground squirrels, pocket gophers, prairie dogs and jack rabbits etc.
- Aluminum phosphide, Zinc phosphide highly toxic in acute exposures, it converted to phosphine gas by the moisture and acidity of the stomach.
- **Silver nitrate test** is performed to analyze the presence of phosphides. In zinc phosphide poisoning quantitative estimation is done by using **Trace metal analyzer.**
- **Cow dung powder** is the second commonest acute poisoning cases for the hospital admission.
- Cow dung powder is available in two different colors: yellow powder (Auramine O) and green powder (Malachite Green) commonly used in rural Tamilnadu (South India) in the districts of Coimbatore, Erode and Tirupur. Even though the sale is legally banned, the powder is easily available in grocery shops. It can cause gastrointestinal symptoms and persistent seizures sometimes.
- Pattern of poisoning in a region depends upon various factors such as availability, cost and access to toxic agents, socioeconomic status, cultural and religious characteristics of people.
- Cow dung powder has been so widely used, that the district authorities banned the sale of this product in 2007. However, it is still widely available and there is no trend of a decrease in the incidence of cow dung powder poisoning during the study period. This underscores the fact that banning such substances, without educating the public or tackling the fundamental cause of deliberate self-harm, will not succeed.
- Apart from the hematological and biochemical tests toxicology departments offers qualitative and quantitative analytical methods.
- Most of the agricultural poisoning shows variations in level of SGPT, SGOT, RFT, creatinine, urea, total bilirubin, etc.
- 164 testes are performing in the analytical toxicology departments. Specific test include drug abuse, toxicological screening, metal screening, water portabilityusing specific standards, therapeutic drug monitoring, quantification of poisons, individual

toxin analysis, identification of adulterants, toxins etc.. From commercially available food products.

- Analytical toxicology will help the detection, identification, and often also the measurement of other drugs and other foreign compounds (xenobiotic) in biological and related specimens to help in the diagnosis, treatment, Prognosis and prevention of poisoning.

CONCLUSION

From our study it was revealed that,

- Agricultural poisons were found to be the most common cause of acute poisoning (55%).
- The most common mode of poisoning was suicidal (69%) and followed by accidental poisoning cases (31%).
- Most of poisoning patients was full recovered with or without specific treatment (81%).
- Accidental poisoning was mostly including animal envenomation's (28%).
- Males were the major victims (61%).
- Most poisoning cases are reported from rural areas (70%).
- Most of the treatments were provided to the patients according to symptomatic treatment only, based on the evidence or sample of poisons brought with patients.

FUTURE DIRECTION

The present study mainly focussed and analysed with variations in treatment methods of different kind of poisons. Based on the result, I concluded that the agricultural poisons cases were the most common causes of acute poisoning, especially in rural area.

Further more, the quantitative analysis of estimation of active poison metabolite from our body fluid will helpful for further recovery of the poisons victims. Even though, we could avoid the 21% occurrence of mortality as per our project report in tertiary care hospitals located at Erode & Tiripure region of Tamil Nadu.

In addition to what we proceeds, due to short period of study time and also low economic back ground of our poisonous victims, it was not able to perform quantitative estimation.

So, we strongly recommended to carry out quantitative analysis to the future researches to this relevant topic will be helpful for 100% recovery in very near future.

BIBLIOGRAPHY

1. Pillay VV. ***Text Book of Forensic Medicine and Toxicology***, 14th edition. Paras publication Mumbai, India, 2004.
2. Dash S K,Mohanty M K. Sociodemographic profile of poisoning cases. ***Journal of Indian Academy of Forensic Medicine***, 2005; 133-138.
3. World Health organization. ***Guidelines for Poison Control Bulletin***2013; Geneva: World health organization.
4. World Health organization. ***Guidelines for Poison Control Bulletin*** 1999; Geneva: World health organization.
5. Yang CC,Wu JF, Ong HC, Hung SC, Kuo YP, Sa CH. Taiwan national poison center epidemiological data 1985-1993**.** ***Journal of Toxicology and Clinical Toxicology.*** 1996
6. Sharma B K, Harish D, Sharma V, Vij K. The epidemiology of poisoning; Anindian view point,***Journal of Forensic Medicine and Toxicology.*** 2002; 5-11.
7. Petridoue, Polychrionopoulou A, Kouri N, Karpathios T, Koussouri M, MessaritakisY .Unintentional childhood poisoning in Athens: a mirror of consumerism ***Clinical Toxicology*** 1997 ; 669-675.
8. Ramesha K N, Moorthy K, Rao B H, Kumar G, Pattern and outcome of acute poisoning cases in a teritiary care hospital in Karnataka,India. ***Indian Journal Of Critical Care Medicine.***2009, 152-155.
9. Singh S, Sharma B K, Wahi P I, Anand B S, Chugh K S.Spectrum of acute poisoning in adults (10 year experiences***). Journal of Associate Physician India.***1984; 561-563**.**
10. Virendar P S, Sharma B R, Dasari H, Krishnan V. A ten year study of acute poisoning cases in teritiary care hospital**.** ***Indian Internet Journal of Forensic Medicine & Toxicology***. 2004
11. Srivasthava A, Peshin SS, Kaleekal T, Gupta SK, An epidemiological study of poisoning cases reported to the national poison information center, all India institute of medical Sciences,New Delhi. ***Human Experimental Toxicology*** 2005; 279-285.
12. Thomas W F,John H D,William R H. ***Stedman's Medical Dictionary***. 28th edition. Lippincott William &wilkins, Newyork (2007).
13. *Dr. Indu A. Chadha* ***Indian Journal Of PoisoningAnaesth****. 2003; 47 (5) : 402-411.*
14. Haddad (1998).The emergency management of poisoning.***Journal Of Emergency Medicine***841-842.

15. JayaprakashH,SaralaN,Venkatarathnamma P N, Kumar T N. Analysis of different types of poisoning in a teritiary car hospital in rural south india**.** ***Journal Of Food Chemical Toxicology***.2011;248-250.

16. Emerson G.N, Sharma R K,Dogra T D. Analysis of poisonous plants in India. ***Journal of Forensic Medicine and Toxicology.*** 1999; 21-25.

17. Aggarwal P, Common poisoning in india. ***Forensic Medicine*** 1998. 73-75.

18. Cruz LS, Vargas R, Lopes AA. Snakebite envenomation and death in the developing world. ***Medical Journal (MARS)*** 2009.

19. ChippauxJP,Alag N A, Envenomation and poisoning by venomous or poisonous animals. ***Medical Journal(MARS***) 2008; 215-221.

20. Josef O. Fadare, Afolabi OA management of snake bite in resource challenged settings:Arevew of 18 months experience in a Nigerian hospital ***Journal Of Clinical Medicine And Research Vol.***4(3), PP. 39-43, MARCH 2012

21. *Dr. Indu A. Chadha****Indian Journal Of PoisoningAnaesth****. 2003; 47 (5) : 402-411*

22. Warrell D.A WHO/SERO Guidelines for the clinical management of snake bit in the Southeast Asian region. ***SE Asian J Trop Med Pub Health***. 200; 11:1-85.

23. .National snake bit management protocol, india.(2008) [online] available at ***www:// mohfw.inc.in*** (directory general of health and family welfare, ministry of health and family welfare, India). Search on (2-3-2016)

24. BosnakM,E, Yolbas I, Bosnak V, Kapian M, Gurkan F, Scorpion sting envenomation in children in south east turkey. ***Wilderness Environmental Medical Journal*** 2009; 118-124.

25. Aaron R, Joseph A, Abraham S, Muliyil J, George K, Fundamental management of poisoning ***Lancet Journal Of India***. 2004; 1117- 1118.

26. Mathew J. Ellenhorn, MD, Los Angeles, CA Donald G, diagnosis and treatment of human poisoning, ***British Medical Journel***. 2007: 8-108

27. Adalkha A, Philip P j, Dhar K L. Organophosphorus and carbamatepoisoningin Punjab, ***Journal Of Associate Physicianindia*** 1986; 36:210

28. Bajaj R, Wasir H S, Epidemic of Aluminium phosphide poisoning in northern india.***Lancet.*** 1988 ;11:820

29. Gargi J, Tejpal H R. A Retrospective autopsy study of poisoning in the northern region of Punjab. ***Journal of Punjab academy of forensic medicine and Toxicology***.2008; 17-20.

30. Das R K .Epidemiology of insecticide poisoning at A.I.I.M.S Emergency service and role of its detection by gas liquid chromatography in diagnosis. ***Medicolegal update***2007; 49-60.

31. AS Jaswal, S. Narayan.simultaneous determination of zinc (Zn), cadmium (Cd), lead (Pb) copper (Cu) in blood using differential - pulse ***Journal Of Cellular Biochemistry*** 2004; 44-67.

32. Tsakallof AK, Margariti MG. the analytical method of biological monitoring for exposure to pesticides ***The Drug Monitoring*** 2007 April 29(2);150-163.

33. Brady W.J, Ma G, Pollack M, Chan T.C. Electrocardiographic manifestation: digitalis. ***Journal Of Emergency Medication***.2001 February 20(2);145-152**.**

34. A. Wahaba, S. Wahaba, M.S Zaheer, R.A Khan. Acute aluminum phosphide poisoning: An update, ***Hong Kong Journel of Emergency Medicine.***2008;15:152-155.

35. Ryan A.Stephen, Sheehan W.Donal, O.Neill, B.Sobhain and Costello S.Danial. Carbone monoxide poisoning: novel magnetic resonance imaging patern. ***International Journel Of Emergency Medication,*** 2012 June 28;5-30.

36. Hong-Tao Zhang, MD, Bao-Ping Qiao, MD, Bao-Ping Liu, MS and Xian-Guo Zhao, MD. Study on the Treatment of Acute Thallium Poisoning ***The American Journal of the Medical Sciences*** Volume 347, Number 5, May 2014 .

37. Hong-Xiang Liu*, Chang-Fang Liu, Wan-Hua Yang Clinical study of continuous micropump infusion of atropine and pralidoxime chloride for treatment of severe acute organophosphorus insecticide poisoning ***journal of the Chinese Medical Association*** 78 (2015) 709e713.

38. O. Zubiaur, J. Salazar, B. Azkunaga, S. Mintegi∗, Grupo de Trabajo Therapeutic psychotropic drugs: Most common causeof unintentional poisoning inchildren (2014) ***www.anlalesdepediatria.org An Pediatr (Barc).*** 2015; 83:244-247.

39. M.Mangaraj, P.K.Mishra, E.Venkata Rao Laboratory Abnormalities in Patients with Organophosphorous Poisoning, ***Indian Medical Gazette,*** januvary 2014:6-12.

40. Shilpa N. Dhotre, Raghavendra V. Katkam, Nitin G. Joshi, Kishor H. Deshpande Study of Acetyl Cholinesterase, ButyrylCholinestrase and β - Glucuronidase in Organophosphorus Poisoning ***Indian medical gazette*** (2014);51-57.

41. S. Panda, R. Nanda. Laboratory Abnormalities in Patients with Organophosphorous Poisoning ***Indiam Medical Gezette***jan (2014);37-44.

42. Supreeti Biswas, MithileshHaldar, Kanchan Kumar Mondal, Chanchal Kumar Dalai, Swati Bhattacharyya, Saptarshi Chatterjee, Anup Kumar DasRationally Used Antidotes in Organophosphorus Poisoning Prevents Suicidal Death, ***Journal of Indian Academic Forensic Medicine.*** Jan-March 2013, Vol. 35, No.1;51-54.

43. Chandra ShekharWaghmare, Manish Nigam, Pradeep K Mishra, Shailendra PatelThe Study of Investigational Profile of Common Pesticide Poisoning Cases admitted at SAIMS, Indore. ***Journal of Indian Academic Forensic Medicine***. January-March 2014, Vol. 36, No. 1

44. Surendra Khosya1* and SR Meena2Current Trends of Poisoning: An Experience at a Tertiary Care Hospital Hadoti Region, Rajasthan**.** ***Indian Journal of Clinical Toxicology*** (2016) 6:2.

45. Amarnath Mishra1, Shukla S.K, Yadav M.K And Gupta A.k: Epidemiological Study of Medico legal Organophosphorus Poisoning in Central Region of Nepal .***Journal Of Forensic Research***(2012); 3:9-18.

46. Dr.Sanjeevkumar Chaudhary1, Dr. S.G. Momin2 Dr.Dipak H. Vora3, Dr.Pikesh Modi4, Dr. Viral Chauhan5 , Dr.Dipak Chotaliya5;An Epidemiological Study of Fatal Aluminium Phosphide Poisoning At Rajkot ***IOSR Journal of Pharmacy*** e-ISSN: 2250-3013, p-ISSN: 2319-4219, www.iosrphr.org Vol. 3, Issue 1, Jan.-Feb (2013), PP. 17-23.

A STUDY OF OBSERVATION IN TREATMENT MODALITY OF ACUTE AND CHRONIC POISONING CASES BY ANALYSING CLINICAL LABORATORY PARAMETERS AND ITS OUTCOME IN TERTIARY CARE HOSPITALS.

PATIENT PROFORMA

NAME: **IP NO:** **AGE:**

SEX: **HP NO:**

MB NO:

DOA: **DOA:**

BLOOD GROUP:

DEPARTMENT:

NAME OF PATIENT CARE CANDIDATES:

COMMUNICATION ADDRESS:

CHIEF SYMPTOMS:

CHIEF COMPLAINTS:

PATIENT HISTORY:

DIAGNOSIS:

DISCHARGE SUMMARY

VITAL EXAMINATION:

BP:

PULSE:

R RATE:

TREATMENT:

RADIOLOGY:

ELECTROCARDIOGRAPHY REPORT;

ECHOCARDIOGRAPHY REPORT;

INVESTIGATION:

COURSE TO HOSPITAL:

ADVISE ON DISCHARGE [DIETING]:

ADVICE ON DISCHARGE [DISCHARGE MEDICATION]:

S.NO.	NAME OF DRUG	DOSE	ROUTE	FREQUENCY

PAST MEDICATIION HISTORY:

SINO	NAME OF DRUG	DOSE	ROUTE	FREQ

PERSONAL HISTORY:

SOCIAL HISTORY:

PHYSICAL EXAMINATION:

DATE	TEMP	BP	PULSE	RR

LABORATORY PARAMETERS

Tests	Date & parameters																				
CHOLINESTRASE																					
UREA																					
GRF																					
CREATININE																					
SODIUM																					
POTASSIUM																					
BICARBONATE																					
MAGNESIUM																					
CHLORIDE																					
PHOSPHOROUS																					
CALCIUM																					
Hb																					
CT																					
WBCT																					
TC																					
PH																					
PCT																					
CRP																					
BLOOD &BD PCT																					
HD																					

INTAKE																					
OUT PUT																					
WIGHT																					
AMYLASE																					
LIPASE																					
ALBUMIN																					
GLOBULIN																					
S GOT																					
S GPT																					
GAMA GT																					
ALB/GLO R																					
ALK PHOS																					
TP																					
TB																					
DB																					
IB																					
ACT																					
ESR																					
CK																					
CK MB																					

TREATMENT AGAINST SIMPTOMS

DRUG	DOSE	ROUTE	FREQUENCY	DATE																		

LABORATORY INVESTIGATION:

<table>
<tr><td colspan="4">DEPARTMENT OF ANALYTICAL TOXICOLOGY

TOXICOLOGY WOKSHEET</td><td>QUALITATIVE ANALYSIS</td><td>LABORATORY NO</td><td>TEST PERFORMED</td></tr>
<tr><td colspan="4" rowspan="5">PATIENT:

DOCTOR:

HOSPITAL:

TELEPHONE NO:

ASSAY REQUESTED:

PRIORITY:</td><td>1)Salicylates</td><td></td><td></td></tr>
<tr><td>2)Phenothiazide</td><td></td><td></td></tr>
<tr><td>3)Paracetamol</td><td></td><td></td></tr>
<tr><td>4)Trichloro compounds</td><td></td><td></td></tr>
<tr><td>5)Paraquate /diquat</td><td></td><td></td></tr>
<tr><td>Sample Type</td><td>Laboratory no.</td><td>Date</td><td>Time</td><td>6)Ethanol</td><td></td><td></td></tr>
<tr><td rowspan="10"></td><td rowspan="10"></td><td rowspan="10"></td><td rowspan="10"></td><td>7)Iron</td><td></td><td></td></tr>
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<tr><td>9)Chlorates</td><td></td><td></td></tr>
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Date: **Signature:**

Address:

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